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A MANUAL
OF
DISEASES OF THE NERVOUS SYSTEM

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OF
DISEASES OF THE NERVOUS SYSTEM

BY
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VOLUME I
DISEASES OF THE SPINAL CORD AND NERVES



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P R E F A C E

THIS volume contains the first part of an attempt to give an account of diseases of the nervous system, sufficiently concise to be within the compass of the time-pressed student or busy practitioner, and yet adequate in its outline of a subject which has become wide and deep beyond any other part of medicine. Success in both aims can scarcely be more than approximate.

Most of the illustrations are printed from blocks prepared from original drawings by phototype processes, and I am indebted to Messrs. A. and W. Dawson, of the Typographic Etching Company (by whom most of the work has been done) for the care they have taken in the reproduction.

The casual reader may perhaps miss subsidiary letters in the illustrations of the lesions of the spinal cord. The omission of these is intentional. A knowledge of the structural topography of the cord is the first requisite in the study of its morbid anatomy, and when this knowledge is gained, the figures will be understood without difficulty. Familiarity with unlettered illustrations facilitates the comprehension of sections of the spinal cord.

By an oversight I have omitted to state that Fig. 84 is copied from Leyden.

In addition to the obligations acknowledged in the course of the book, I have to express my special thanks to Dr. Money for various help, including a perusal of the proof-sheets and the preparation of the index and table of contents.

QUEEN ANNE STREET, LONDON ;
August, 1886.

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DISEASES OF THE NERVOUS SYSTEM.

INTRODUCTION.

CLASSIFICATION OF DISEASES OF THE NERVOUS SYSTEM.

A CLASSIFICATION of Diseases of the Nervous System, at once scientific, exact, and convenient for systematic description, is not at present possible. If we attempt to classify the diseases according to either seat or nature, we are met at once by difficulties which prevent any complete arrangement. These difficulties arise from the facts that many diseases affect more than one part of the nervous system, [that the precise seat of other maladies is unknown, and that we are still ignorant of the nature of a considerable number of diseases. A rough division is often made into two broad classes of "organic" and "functional" disease. The first class of "organic diseases" comprehends those in which there is always a visible lesion, manifesting the morbid process which constitutes the disease. The second class of "functional diseases" is less definite, comprehending (1) those diseases that consist only in a disturbance of function, and are therefore properly so designated, and (2) many diseases which have this in common with true functional disease that they are transient and not permanent, and that they are not known to depend on organic changes. But there is also a large class of diseases in which no constant lesion has been discovered, but which are not transient, and cannot well be placed in either of these classes. This simple classification is therefore inadequate. It may be well to consider in what respect it needs alteration to meet the requirements of our present knowledge.

The term organic disease, as commonly used, means visible disease. But visibility is partly relative; it depends on the means of vision. The application of the microscope to pathology immediately increased the range of organic disease, and a similar increase has resulted, in our own day, from the use of staining agents, which render visible changes that could not before be seen. Still it is the degree of alteration that determines visibility. Molecular changes in nutrition, considered

as such, must be colossal to be detected. Such alterations, not sufficient to be seen, but still considerable, probably constitute the morbid process in many diseases that are commonly classed as functional. Hence we may distinguish a class of "nutritional diseases" from those that consist in a mere derangement of function. While distinguishing these two classes, we cannot entirely separate them, because nutrition and function are mutually dependent, and prolonged functional disturbance may determine nutritional change.

Visible disease varies much in nature and degree. Some forms can always be seen with the unassisted eye; they change the shape, or colour, or consistence of the part. Other forms can be detected only with the aid of the microscope, or may be seen with the naked eye when considerable in degree, and only through the microscope when slight in degree. It is convenient to term these "structural diseases." Thus we may distinguish four pathological classes of disease of the nervous system:

1. *Organic disease* (or *coarse organic disease*); such as tumour, hæmorrhage, softening.
2. *Structural disease*; such as most forms of sclerosis.
3. *Nutritional disease*; such as general paralysis of the insane, paralysis agitans, chorea.
4. *Functional disease*; such as reflex convulsions, and many forms of hysteria.

In the first class the morbid process always begins outside the nerve-elements themselves. In the second class it may begin within or outside them. In the third and fourth classes these elements are probably always primarily affected.

Such a classification helps us to obtain clearer views of the primary relations of disease, but is not convenient for systematic description. For this a hybrid system is necessary, in which organic and structural diseases are classified according to their seat,—in the nerves, spinal cord, or brain. With each group may be placed some nutritional or functional maladies of local incidence. The bulk of such diseases are best considered after the organic diseases have been described, since many of them are of wide distribution or uncertain seat.

PART I.

GENERAL SYMPTOMATOLOGY.

SYMPTOMS AND THEIR INVESTIGATION.

THE nervous system is almost entirely inaccessible to direct examination. The exceptions to this are trifling. The termination of one nerve, the optic, can be seen within the eye. Some of the nerve-trunks in the limbs can be felt; a few, as the ulnar, in the normal state; others only when enlarged by disease. As a rule, morbid states of the nervous system can be recognised only by the derangement of function that they cause. The functions of the nervous system are often roughly divided into mind, motion, and sensation, but they are far more extensive than those words denote. The functions of all the organs of the body, all states of vessel and of circulation, and all processes of nutrition, are under the control of the nervous system. The disturbance of nerve function produced by disease is conterminous in extent with the organism itself. Our knowledge of these symptoms is constantly increasing in range and in degree. They differ, however, very much in relative importance. Some are more frequent in occurrence than others, and are more direct in significance. Many have a special relation to certain parts of the nervous system, and may be considered most conveniently when we deal with the diseases of that part. Others occur in diseases of all parts. It may be well, at the outset, briefly to describe the general characters of some of these most frequent symptoms.

MOTOR SYMPTOMS.—Disturbance of motion is one of the most common of the effects of disease. Movement is produced by muscles, but the muscles are excited to contraction by the nervous system. Most alterations of motility, both defect and excess, are due to nerve derangement. The conditions are very few in which motor changes originate in the muscles themselves. Defect of movement is of muscular origin only in some cases of primary alteration in the nutrition of the muscles. When there is a general impairment of nutrition, the muscles participate in this; they are ill-nourished, and

therefore feeble. Such a condition is met with in chronic wasting diseases, in anæmia, and as an acute affection in all acute febrile diseases. This is the chief cause of the muscular weakness that so often succeeds fevers. In some chronic diseases the muscles undergo a primary degeneration. One is an extremely rare form of muscular atrophy. Another is the disease known as "pseudo-hypertrophic paralysis." A third is ossification of the muscles. In almost all other conditions, loss of power in the muscles is dependent on the state of the nervous system.

So also with increased and involuntary muscular action—spasm. We know even less of what has been termed "idio-muscular contraction," that is, contraction originating in the muscle itself, than we do of "idio-muscular paralysis." Whenever the whole of a muscle contracts, the contraction is probably always due to the influence of the nervous system, even though, as in ordinary cramp, it seems to us to be purely local. We can scarcely conceive that all the fibres of a muscle can contract simultaneously except under central influence. Even muscular "tone" is dependent on the connection of the muscle with the nerve-centre. The partial contractions that are termed "fibrillary" are perhaps local, since they are readily produced by mechanical stimulation, such as a tap on the muscle. But it is probable that fibrillary contractions, although sometimes local, are often of central origin. This is true also of the flickering contractions that are so common in the orbicularis palpebrarum and other muscles, in persons whose nervous system is enfeebled.

Defect of movement may present every degree, from slight weakness to absolute loss. There may be an inability even to make the muscle contract, or the voluntary contraction may be too feeble to move the parts to which the muscle is attached. This difference is to some extent relative, since it varies according to the mobility of the part to be moved. A contraction that would move a finger may be insufficient to move a leg. The difference may vary in the same part according to position. A degree of power in the flexors of the hip that will move the leg when the patient is lying on his side may be insufficient to do so if he is lying on the back. Or the part may be moved, but with less force than normal. The term "paralysis" has long been applied to all degrees of defect of power, a distinction being made between complete and incomplete paralysis. Partial loss of power is also sometimes termed "paresis."

Our means of measuring defects of muscular power are very imperfect. The force of some movements can be measured by means of an instrument called a "dynamometer." Several forms of dynamometer have been devised, but that in general use consists of an oval steel ring, which can be narrowed either by compression applied to its smaller diameter, or by traction applied to its longer diameter. The change in the shape of the dynamometer, and therefore the force exerted, is shown by the movement of an index on a scale that has a

double graduation, an inner for compression, and an outer for traction. This form of dynamometer is known as "Charrière's" from the name of its first maker. An older form of dynamometer is that designed by Duchenne. It is less convenient for testing the grasp, although better adapted for indicating the force of traction. Practically, however, the use of these instruments is confined to the measurement of the grasp. The estimation of the strength of other movements is difficult, and liable to much error. In examining these it is best for the observer to oppose the movement and note the resistance that the patient can overcome. The wide variations in strength among different persons renders absolute estimation possible only when the defect is considerable. Slight changes are recognised by comparing the power on the two sides. It should be remembered that the limbs on the right side are normally stronger than those on the left, the relation being about as five to four. Care must be taken to secure uniformity of the conditions under which the observations are made.

Inco-ordination of Movement.—A muscular movement involves an exactly proportioned contraction in the muscles that effect the movement, often numerous, and co-operating in various degrees. It involves also a contraction in the opponents of these muscles, less in degree, but with equal delicacy of proportioned strength. If, instead of this balanced adjustment, the contraction varies in degree in the same or in different muscles, the movement is not that which is intended—is irregular. An excess or deficiency of contraction leads to a corresponding change in the contracting opponents. Attempts to correct the error may themselves be wrong in degree and increase the disturbance. This condition is termed inco-ordination or "ataxy." It may be simple unsteadiness of movement, or the excessive contractions may be so sudden and considerable as to cause "jerky inco-ordination." It is readily recognised by simple observation, or may be recorded by an attempt to draw a straight line on a sheet of paper, or to hold a point in contact with a moving surface (*e.g.* of blackened paper) on which the irregularities of movement are shown.

Another method of obtaining evidence of it has been suggested by Blix. It consists in making the patient suddenly try to touch with the point of a pencil a spot on a sheet of paper suspended before him, his eyes being closed at the moment of the attempt. After a certain number of attempts the average distance from the spot of the dots he has made is ascertained, and, compared with the result given by a normal individual, affords a numerical indication of the degree of inco-ordination. The method is effective, although the result is scarcely worth the trouble.*

Whenever there is much inco-ordination, spontaneous movements occur when the patient tries to hold the limb still in a certain posture. The irregularity obtains in the fixed as well as in the mobile condition. In the latter it causes ataxy, in the former spontaneous movements.

* Blix, 'Neur. Centralb.,' 1884, p. 83.

SENSORY SYMPTOMS.—Deranged sensation is another very common symptom. It has often to be searched for, as a significant defect may be unknown to the patient. Each form of sensation, of touch, temperature, and pain, must be separately tested, since one may be affected, and not another.

Tactile Sensibility.—In testing tactile sensibility care must be taken that the instrument employed does not give an impression of heat or cold, lest the patient perceive by the sense of temperature that which he cannot discern by the sense of touch. The observer's finger may be used, if it is not cold, or the skin may be touched with the feather end of a quill pen. The eyes of the patient should be closed during the examination, and, since he is apt to fancy that he feels a light touch when he does not, he should occasionally be asked if he can feel, when no contact is made. If there is no absolute loss, the character of the sensation may be changed; throughout one side a touch may cause a sensation distinctly different from that which is produced when the other side is touched. Loss of sensibility to touch is termed "anæsthesia," but this word is often applied loosely to all forms of impaired sensibility.

In examining the tactile sensibility, it is important to ascertain, not only whether the patient can feel, but whether he is able to recognise the place touched,—whether he can correctly "localise" the sensation. For this he must be asked, not only whether he feels the touch, but to say or point out where he feels it. The part touched should be frequently varied, and the eyes, of course, kept closed. Another test for tactile sensibility depends on the fact, ascertained by E. H. Weber, that the distance apart at which two points are discriminated is nearly the same in different individuals in the same part of the body, although it varies much in different parts of the body. The normal distance being known, the increased distance at which the points have to be placed for their discrimination is a measure of the degree of defect. The points should not be so sharp as to occasion pain. A pair of blunt-pointed compasses may be employed, but the most convenient instrument is one contrived by Dr. Sieveking, and called the "æsthesiometer." It consists of a graduated bar, and two points, one of them being moveable and the other fixed. The greater the degree of tactile sensibility, the nearer together can the points be, and still be discriminated. The distance at which they are normally distinguished must be known before any inference can be drawn. The most important average distances, ascertained by Weber, are as follows: the distance is smallest, *i. e.* the sensitiveness is greatest, at the tip of the tongue, where the points are discriminated when only 1.5 mm. apart. Then come, in order of sensitiveness, the finger-tips, 2 to 3 mm.; the lips, 4 to 5 mm.; the tip of the nose, 6 mm.; the cheeks and the backs of the fingers, about 12 mm.; the forehead, 22 mm.; the neck 34 mm.; the forearm, lower leg, and back of foot, 40 mm.; the chest 45 mm.; the back, 60 mm.; the upper arm and thigh 75 mm. Slight variations

exist in different individuals, and a deviation from the normal that is uniform in degree throughout the body is probably physiological. In using this test it is necessary to touch the skin with the two points at the same moment, and with equal pressure. The examination requires time and patience, and the results are considerably modified by the intelligence of the patient. Moreover, the power of discrimination is increased by practice. It is rare to obtain a conclusive result unless there is a degree of defect that causes a slight absolute loss, such as may be detected with a feather. For these reasons, the practical value of the test is less than was at first anticipated. It is more useful for the estimation of changes of sensibility in the same person and the same part than for actual diagnosis.

Another test that has been proposed is to move a pointed object along the skin, and note the minimum movement that can be recognised as such (Leube). This test has not come into general use.

Curious modifications of tactile sensibility are sometimes observed. In one of these, a single touch is felt as if it were two or even three, a condition that has been termed "polyæsthesia" (Fischer). In another, an impression on one part is referred to some other part, usually in the same limb, rarely in another limb. In another variety, an impression on one part has been referred to the corresponding place on the opposite side of the body; this has been termed "allocheiria" (Obersteiner).

Perception of the degree of pressure on the skin is probably subserved by the nerves for tactile sensibility. It may be estimated by simple pressure applied through any instrument, or by small weights, or by an instrument contrived for measuring the pressure applied to an artery to ascertain the tension of the pulse. In this, a spring indicates the degree of pressure applied. The part tested must be supported, so that the muscles are not brought into action. The point to be ascertained is the minimum variation that can be recognised. In health, this is about one twentieth of the total pressure, whatever the latter may be.

Sensibility to pain is subserved by what are called nerves of "common sensibility." It may be tested by a prick or a pinch. For a prick, too fine a point must not be used, not only because a sharp point may inflict a needless wound, but because, in the less sensitive parts of the skin, where the terminal nerve-plexus is wide, a fine point may here and there be unfelt, although it penetrates the skin. Hence a somewhat blunt point should be employed. Nothing answers better than the point of a quill pen. The faradaic current may be employed to test the sensibility to pain, wire terminals being most suitable for the purpose. Its advantage is the delicacy with which it can be graduated, but it does not furnish any absolute standard, and is chiefly useful for comparing the sensitiveness of corresponding regions on the two sides.

Sensibility to pain may be changed with or without sensibility to touch. Its loss is termed "analgesia," but is often included in the general term "anæsthesia." A painful sensation is often felt more acutely than normal; this is called "hyperæsthesia," or, more correctly "hyperalgesia." Occasionally a touch on the skin gives rise to pain, but it is probable that this is due to the stimulation of the over-sensitive nerves of common sensibility, and is not an intensification of a tactile sensation. Both tactile and painful impressions may produce sensations that are abnormal in character, described as "thrilling," "tingling," &c. This perverted sensation has been termed "paræsthesia," or "dysæsthesia," words that have also been applied to purely subjective sensations.

Sensibility to temperature is usually affected with sensibility to pain, but often not in the same degree, and one may be impaired without the other. There may be an absolute inability to recognise either heat or cold as such, or slight degrees of each may be unperceived, while considerable degrees are recognised correctly. In the latter case there is impairment of the power of perceiving differences in temperature, analogous to the defect in the perception of differences in pressure. There may be a perverse sensation, whereby hot objects feel cold, and cold objects hot. The pain that extreme degrees of temperature normally produce may be felt with undue readiness or less readily than normal.* When perception of pain is delayed, that of temperature may be delayed also. It must be remembered that, in health, this sensation is less rapid than that of pain, because time is required to raise the temperature of the skin sufficiently to stimulate its nerves. For coarse examination, hot and cold spoons may be employed; for ascertaining the power of differential discrimination, test-tubes, containing water at different temperatures, are necessary. For practical purposes, the result thus obtained is hardly commensurate with the time required for the examination.

It is necessary always to test separately the sensibility to heat and to cold, as there is strong reason to believe that these are subserved by different nerves. If very minute points of the skin are examined, it is found that at some points only heat, at others only cold, is perceived. The stimulation of points of the skin by electricity shows also that at some points the peculiar electrical sensation is produced, at others, sensation of cold, at others sensation of heat. Further, the points identified as "cold points" and "warm points" by the one method, correspond with the points ascertained by the other method.† The conclusion from these observations is confirmed by the fact that in disease the sensibility to heat and to cold may be affected in different degrees.

* See on this point Donath, 'Arch. f. Psych.,' Bd. xv.

† Blix, 'Zeitschr. f. Biologie,' Bd. xx, p. 141; Eulenberg, 'Zeitschr. f. klin. Med.,' Bd. ix, Heft ii. The observations have also been corroborated by Goldscheider and Herzen.

The temperature sense varies much in different parts. Sensibility to cold is least at the epigastrium, that to heat least on the back. In the leg, sensitiveness to both heat and cold is greatest at the knee, and lessens upwards and downwards.

Muscular Sensibility.—The term “muscular sense,” as often used, includes more than one form of sensation. There is, first, a true muscular sensibility. The muscles are abundantly supplied with sensory nerves, which are stimulated by pressure and by extension. They are said to end in the interstitial tissue between the muscular fibres (Tschirjew). A painful sensation is readily produced by compression of a muscle, and the pain of cramp, distinctly felt in the substance of the muscle, is probably due to the compression of these nerves by the extreme shortening (and therefore widening) of the muscular fibres in their contraction. After cramp, the nerves are left in a state of exalted excitability, so that extension of the muscle gives rise to pain that is not produced in the normal state. The pain caused by a strong electrical stimulation is probably due to the excitation of these nerves, either direct, or by their compression. They appear to subserve the consciousness of contraction which is involved in the recognition of resistance (such as the estimation of a weight), and also, in part, the recognition of posture, even on passive movement. The muscles adapt themselves to all passive movements, and the impression of the elongation or contraction of the muscle probably enters, with the impressions from the skin, &c., into the perception of posture. When the limb is at rest, sensory impressions from the skin and joints probably constitute the chief source of information.

In some morbid conditions the muscular sense is lost, the patient cannot appreciate the difference between light and heavy objects. A poker and a feather seem to be of the same weight. He is also unable to recognise the posture into which his limbs are put by another person. This loss may occur when tactile sensibility is unimpaired. Conversely, the sensibility of the skin may be lost, while that of the muscles is unaffected. This separate affection of cutaneous and muscular sensibility is often seen produced by unilateral lesions of the spinal cord. This question is further considered in the account of the conducting paths of the cord.

The chief tests for the muscular sensibility are the following:—(1) The power of appreciating differences of weight, *i. e.* variations in the resistance to contraction. In this examination the patient's eyes should be closed, and the objects used should be of uniform size. Leather balls, like cricket balls, containing various weights, are in use at the Queen's Square Hospital for this purpose. It is desirable to reduce the stimulation of the cutaneous nerves to a minimum, by placing the weights in a bag suspended by a string to the part to be tested so that only a small area of the skin shall be pressed upon. The form of the weight is then of little importance. The point to be tested is the power of discriminating differences in weight. This is

greater than in the case of the nerves of the skin, since a difference of one fortieth of the total weight can be recognised under normal conditions. (2) The power of recognising the posture into which the limb is put; if cutaneous sensibility is normal the parts handled must be grasped firmly, and pressed on both sides, so that the direction of pressure may not suggest the posture. Several observations should be made to eliminate the chance of accidental correctness. (3) The sensitiveness of the muscles to deep pressure. (4) Their sensitiveness to electrical stimulation. It is difficult to test accurately the electro-sensibility unless the sensitiveness of the skin is removed, as by the injection of cocain.

Another source of information regarding motor processes is afforded by the degree of central innervation, *i.e.* the activity of the motorcentres. This was pointed out by Sir Charles Bell. It is the innervation, and not the actual muscular contraction, that determines the strength of associated movements, and influences the centre concerned in maintaining the equilibrium of the body. We see this clearly in the case of the muscles of the eyeball. If an ocular muscle is weak, and an increased effort is necessary to direct the eye towards a given object, the object is referred to the place, not towards which the eye is directed, but towards which it would be directed by the increased innervation if the muscle were normally strong. If the patient tries to touch the object, the hand is moved in accordance with the innervation, and goes too far, beyond the object. Hence it is clear that the guide to other motor centres is the central innervation, and not the accomplished movement,—not therefore, the peripheral stimulation of the muscle-nerves. It is equally clear that it is the peripheral stimulation of the muscle-nerves that is concerned in the sense of resistance, because the appreciation of weights is not interfered with by simple weakness. It is doubtful whether the impression of motor innervation affects consciousness so as to constitute an actual sensation, comparable to that due to the other sensory nerves, or even to the muscle-nerves.

REFLEX ACTION.—Many symptoms of disease of the nervous system are due to derangement of the various reflex actions. These are numerous, and we can now only consider their general characters. Each action is effected through an afferent sensory nerve, an efferent motor nerve, and a system of nerve-cells and fibres connecting the two in the spinal cord, the “reflex centre.” The centre is usually a complex and often extensive structure, and in it are paths of different “resistance,” determining the form of reflex action and its extent, according to the source and intensity of the sensory impression. The reflex centre is between the roots of the sensory and motor nerves concerned, and with them constitutes what may be termed a “reflex arc.” The sensory impulse may not only excite a motor process, it may also pass up to the brain, and affect consciousness as a sensation. Further,

the reflex centre is to some extent under the control of the cerebral centres.

Cutaneous Reflex Action.—Two chief forms of reflex action must be distinguished. The first is that excited by stimulation of the skin, more readily by a gentle stimulation, as a touch, than by a strong, painful impression. The cutaneous reflex actions may be excited at almost any part of the skin, but at some parts they are very definite in character, and are distinguished by special names. The most important are the “plantar reflex,” from the sole; the “gluteal reflex,” a contraction in the gluteus when the skin over the muscle is stimulated; the “cremaster reflex,” a retraction of the testicle on stimulation of the skin on the inner part of the thigh; the “abdominal reflex,” in the muscles of the abdominal wall when the skin over the side of the abdomen is stroked; the upper part of this reflex is a very definite contraction at the epigastrium, and has been termed the “epigastric reflex.” A series of reflex actions may be obtained in the muscles of the back, the highest being in the muscles of the scapula. These spinal reflex actions differ in their excitability in different individuals, and are always more readily produced in the young than in the old. In the region of the cranial nerves, the most important reflexes are those of the eye; the conjunctival reflex, the contraction of the iris on exposure of the eye to light, and its dilatation on stimulation of the skin of the neck.

Muscle Reflex Action; “Tendon-Reflexes.”—The second group of phenomena which depend on reflex action are those which have been termed “tendon-reflexes.” They were first systematically studied by Erb and Westphal, but previously partially recognised and employed in diagnosis by Charcot.

The first of these is the jerk of the leg which occurs when the patellar tendon is tapped. It has been called the “knee-phenomenon” by Westphal, the “patellar-tendon-reflex” by Erb, the “knee-jerk” by myself. To obtain the jerk, the knee must be flexed so that the quadriceps femoris is gently extended, and the leg must be free to move. If then the patellar tendon is struck, the quadriceps contracts and jerks the leg forwards. The most convenient position is with the knee to be tested flexed nearly, but not quite, at a right angle. The posture commonly employed is with the leg to be tested across the other, the knee of the supporting leg being at a right angle (Fig. 1). But if the leg to be tested is stout, its tension in this position may be too great to permit of any movement. In such case the observer may place his arm beneath the patient’s thigh, just above the knee, and rest his hand on the patient’s other knee (Fig. 2). Children may sit on the edge of a chair, adults on the edge of a table. If the legs are vertical, the effect of the blow and of the muscular contraction must be carefully distinguished. If the bent fingers of each hand are interlocked, and the hands pulled strongly, the knee-jerk occurs more readily (Jendrassik). It is essential that the muscles should be relaxed from

voluntary contraction. Any contraction of the flexors may be ascertained by feeling the hamstring tendons, and pressure on these by the fingers often helps to secure relaxation. The blow may be given by the side of the hand, a percussion hammer (Fig. 2), or a stethoscope



FIG. 1.—THE KNEE-JERK.

The dotted line indicates the movement which follows the blow on the patellar tendon.



FIG. 2.—THE KNEE-JERK.

Method of obtaining it when it is not readily produced in the ordinary way.

with an india-rubber edge to the ear-piece. If its existence is doubtful, the skin should be bared. In many cases the movement may be obtained by a downward blow upon the patella, by a blow on the quadriceps tendon above the patella, or by a blow on the substance of the muscle, almost as readily and strongly as by a blow on the patellar tendon. In cases in which it is in great pathological excess, it may even be excited by a blow on the tibia. When it is in excess, it may be conveniently brought out in a somewhat different way. As the patient lies in bed, the finger of one hand is placed across the quadriceps tendon just *above* the patella, and the patella pushed down, so as to make the quadriceps tense. The finger is then percussed in the direction in which the patella is being pushed, so as suddenly to increase the tension in the muscle. The blow is instantly followed by a contraction, jerking the patella and finger upwards. Very often this single contraction is immediately succeeded by a second, and this by a third, and so on—a series of quick clonic contractions, “clonus,” recurring as frequently as eight per second. By grasping the patella firmly, and suddenly pushing it downwards, so as to make the muscle tense, this clonus may also be set up, as Erb has shown. It may continue as long as tension is kept up, but instantly ceases when the muscle is relaxed.

The next important phenomenon belonging to this group occurs at the ankle-joint. If the calf muscles, which are connected with the

Achilles tendon, are made tense, and this tendon is tapped, the muscles contract, causing a slight extension movement of the foot; just as the muscles of the thigh contract when the patellar tendon is struck. In cases in which these phenomena are excessive,—just as sudden tension in the thigh-muscles will cause a contraction, followed by others in a continuous series—so, in such cases, if the calf-muscles, which extend the ankle-joint, are suddenly put on the stretch by pressing the hand against the sole of the foot (Fig. 3), a quick contraction occurs, instantly ceasing, but if the pressure is kept up, instantly renewed, and recurring as long as the tension is maintained, as a clonic series of spasmodic contractions—the “ankle-clonus” or “foot-clonus” (or “foot phenomenon”—Westphal). It can often be obtained best when the knee is not completely extended. The movement is very uniform, from six to nine contractions occurring per second. By attaching a writing point to the foot, and making it trace a line on a revolving cylinder covered with blackened paper, tracings may be obtained (Fig. 4), which are almost as regular as the tracings of a



FIG. 3.—METHOD OF ELICITING THE FOOT-CLONUS.

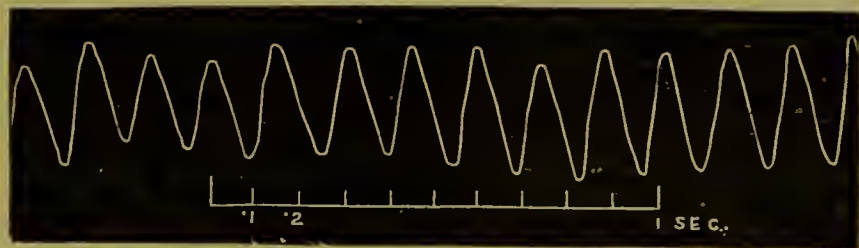


FIG. 4.—TRACING OF THE FOOT-CLONUS IN PARAPLEGIA.
(The tracing reads from right to left.)

tuning-fork. This foot-clonus can be more frequently obtained than the clonus in the extensors of the knee, but the two have the same time and are evidently of the same nature. A clonus quite similar may sometimes be obtained in other muscles—peronei, flexor brevis pollicis, flexors of the fingers, &c.

When a tendon is tapped, and its muscle contracts, the occurrence has somewhat the aspect of a reflex action. It was assumed by Erb that the contraction is a true reflex action, the stimulus being the excitation of nerves in the tendon. Hence it has been termed “tendon-reflex.” This view has received apparent confirmation by the discovery of certain facts: (1) That there are nerves in tendon. (2) That these phenomena depend for their occurrence on the integrity of the

reflex path to, through, and from the spinal cord, and are arrested by a lesion in this path. By experiments on animals (in whom similar contractions may be obtained) it has been found that they are prevented by division of the nerves to the muscles, by division of either the anterior or posterior roots of the spinal nerves, or by destruction of the spinal cord.* The knee-jerk cannot be obtained in locomotor ataxy (damage to the posterior nerve-roots), or in infantile paralysis (damage to the grey matter, the reflex centre). (3) That these phenomena are in excess in some cases, in which the reflex action from the skin is in excess.

These facts certainly prove that some reflex influence is concerned in the production of the phenomena. But (as Westphal has always maintained) they do not necessarily prove that the contractions depend on a simple reflex action from the tendons. Many of the facts cannot be explained by the "tendon-reflex" theory. There is the great fact that passive tension is necessary for the tap on the tendon to be effective, and that when the phenomena are in excess, sudden tension alone will suffice to develop the contraction. But tension acts upon the muscle as well as upon the tendon, and frequently the contraction may be distinctly excited by stimuli which act on the muscle and have no action on the tendon. For instance, in cases in which these phenomena are in excess, if the foot be gently pressed up so as to make the calf-muscles tense, and the muscles on the front of the leg be tapped, the calf-muscles contract (just as they do when, under the same circumstances, the tendon is tapped), and cause a brief extension movement of the foot. I have termed this the "front-tap contraction." It is a very delicate test of increased irritability, and it is also of considerable theoretical interest, since we have in it a contraction developed by a stimulus which does not in any way affect the tendon. It can affect the gastrocnemius directly, for by placing the hand on the calf, a vibration may be felt through the leg. If the tibia, instead of the muscle, is tapped, the contraction is much slighter, or does not occur. Moreover, a tap on the tendon itself only excites the contraction when it increases the tension of the tendon; *i. e.* when it acts upon the muscle also. In the case of the Achilles tendon, this may be easily demonstrated with a little care. A gentle tap on the side of the tendon will excite the contraction as readily as a tap on the back of the tendon, but if the other edge of the tendon is supported (as by the fingers of an assistant), the same tap will no longer be effective, because it no longer increases the tension. The strongest proof, however, of the independence of the phenomena on any stimulation of the tendon is afforded by the experiments of Tschirjew, who divided carefully all the nerves to the patellar tendon, and still found that the tap upon it made the tense muscle contract.

Thus the evidence seems conclusive that the contractions are not excited by stimulation of the nerves of the tendon, but that the stimulus originates in the muscle, the tendon being only, so to speak,

* See Tschirjew, 'Archiv für Psychiatrie,' Bd. viii, Heft 3.

an instrument by which that stimulation is produced. But if the muscle is stimulated and then contracts, is not the contraction excited locally, as Westphal has, from the first, urged? A reflex action takes a certain time, which is needed for the stimulus to travel to and from the cord, and for the reflex process to occur in the centre. According to received physiological data, an interval of at least one fifteenth of a second would be needed for the knee-jerk, if it were a reflex process, and rather more for the movement at the ankle. I have found that when the Achilles tendon, or the front of the leg is tapped, the resulting contraction occurs in about one thirtieth of a second.* The interval for the knee-jerk has been found to be about one twenty-fifth or one thirtieth of a second.† If the patella is pressed down and tapped, in the way I have just described, I have found that the interval between the tap and the resulting contraction is often not more than one fortieth of a second. The shortness of the interval makes it difficult to believe that these contractions can be reflex, and supports the theory that they are excited locally.

But to this view, that the contractions are excited locally, is apparently opposed the fact that they are prevented by whatever lesion arrests reflex action. Some have endeavoured to explain the discrepancy by the suggestion that reflex action may occur in a much shorter time than is commonly supposed. This suggestion is not at present justified by any known facts. Another and, I think, much more probable explanation is this. If we regard the contractions as local, we have still to account for the irritability which permits the local stimulus to cause a contraction. This irritability is developed by passive tension. If the muscle is relaxed, the fibres may contract if they are struck directly, just as do the fibres of a separated frog's muscle, but no contraction can be produced by striking the tendon. Hence I have suggested that the tension excites, by a reflex influence, a state of extreme irritability to local stimulation,—such as that of a tap on the tendon, or such as the vibration from a tap near the muscle, or from a tap on the bone to which the tendon is attached,—which thus excites a visible contraction.‡

The explanation receives some confirmation from the very interesting

* 'Med.-Chir. Trans.,' 1879, p. 292. The measurement has been since confirmed by Waller, 'Brain,' July, 1880.

† .039 sec. Burckhardt; .032—.034 sec. Tschirjew; .04 sec. Brissaud; .03—.04 sec. Waller; .03 sec. Eulenberg. Some measurements which I made of the interval ('Med.-Chir. Trans.,' 1879, p. 275) gave a longer interval, probably in consequence of the movement of the foot being taken as the indication of the commencing contraction. "Load" will increase greatly the period of latent stimulation, probably by causing the initial contraction to expend itself on the elasticity of the muscle. The measurements given above were obtained by recording the commencing contraction of the muscle.

‡ This explanation was originally given for the "foot phenomenon" alone ('Med.-Chir. Trans.,' 1879, p. 295). I now think that it is equally applicable to the knee-jerk. The evidence of the identity in nature of the two is very forcibly stated by Waller (loc. cit.).

observations of Tschirjew.* He has found that if the nerve to a separate muscle be divided, the muscle remains of just the same length. If, however, a weight be first attached to the muscle, when the nerve is divided the muscle lengthens. This shows that the tension does excite a slight contraction which is dependent on a central influence. It is in this condition only that the local stimulation is effective. If the tension put on a muscle is gentle and gradual, it may only develop the irritability, and an additional local stimulation is necessary to produce a visible contraction. If, however, the tension is sudden and forcible, it not only develops the irritability, but produces visible contraction in the muscle thus rendered irritable—as in setting up the foot-clonus. I have shown† that the relaxation of the muscle, between the successive contractions, is not complete: there is a persistent residual contraction, *i.e.* a tonic contraction on which the clonic contractions occur. When one clonic contraction is over, the tension continuing, a second is instantly developed.

The sensory nerves of muscles have been shown by Tschirjew to commence, not within the muscular fibres, but in the interstitial connective tissue. The afferent impulse produced by tension is apparently due to the tension acting on these nerves: the visible contraction is excited by tension or vibration affecting the muscular fibres themselves. The latter is ineffective unless the muscles are brought into a state of special excitability through the cord. The reality of an afferent impulse from the muscle, produced by tension, is demonstrable to anyone who will permit his ankle to be suddenly flexed. A distinct pain is felt in the muscle (none, be it observed, in the tendon). It is not surprising, therefore, that this afferent impulse should, very often, not merely develop the reflex excitability, or tonic contraction, but also cause a more widely-spread reflex action. The attempt to get the foot-clonus, for instance, will cause a flexion of the hip-joint; the attempt to obtain the knee-jerk may cause a movement in the opposite leg or a start back of the body. But these reflex contractions, if carefully observed, confirm the theory which has been put forward in the preceding pages, for they distinctly succeed, at an appreciable interval, the local contraction. Burckhardt has found that the latent interval for a skin-reflex is three times as great as for the knee-jerk. I think that this theory of reflex irritability and local stimulation affords a full explanation of all the relations of these phenomena to the central nervous system, and to the other phenomena of disease, and it is the only theory which adequately explains them.

It seems, therefore, that the term “tendon-reflex” is altogether inaccurate. The phenomena are, according to the explanation above given, dependent on a “muscle-reflex” irritability, which has nothing to do with the tendons. If we wish to describe them by a general term, it is best to employ one which does not involve any special theory of their nature. They may be termed “tendon-muscular

* ‘Reichert und Du Bois-Reymond’s Archiv,’ 1879.

† Loc. cit., p. 286.

phenomena," but the intervention of tendons is not necessary for their production; the one condition which all have in common is that passive tension is essential for their occurrence, and I have suggested* that they be termed *myotatic* contractions (*τατικός*, extended).† The irritability, on which they depend, is due to and demonstrative of a muscle-reflex action which depends on the spinal cord. It is highly probable, as Tschirjew suggested, that the condition on which the myotatic irritability depends, is identical with muscular "tone." Since the experiments of Heidenhain, it has been generally admitted that "tone" depends on tension, and is a reflex process.‡

A true "tendon-reflex" may be excited by pinching the tendon, but this is a start of the whole limb, precisely such as results from a pinch of the skin.

*Paradoxical Muscular Contraction**.—This name has been given by Westphal§ to a slow tonic contraction occurring in a muscle when suddenly relaxed, or rather when its course is suddenly shortened. It is best seen in the tibialis anticus. If the foot is grasped, and passively flexed on the leg, the tibialis anticus contracts, sometimes after an appreciable interval; its tendon stands out, and the contraction keeps the foot flexed for some minutes (in one case for twenty-seven), and then slowly relaxes. Repetition lessens the contraction. It is not voluntary (although a voluntary contraction may simulate it), for it may be unilateral. The contraction produced by faradism or by the will may be similarly lasting. The same phenomenon may be observed in the extensors of the toes, sometimes in the flexors of the knee, rarely in the arm muscles. Its mechanism is uncertain,—whether reflex or not, whether produced through the muscle itself, or through the tension on the antagonists, as Erlenmeyer has maintained. The former is perhaps the more probable. It is interesting as an excess of the adaptation which muscles undergo on their passive elongation or shortening. This is probably one element in the appreciation of posture. In the cases that have presented this phenomenon there has been no muscular rigidity, no great excess (even sometimes a loss) of the myotatic irritability. It occurs sometimes in the early stage of tabes, and in some other affections, but its significance is not known. A phenomenon somewhat similar occurs in hysteria (Féré); the facts of the hysterical cases, and the effects of chloroform observed by Westphal, suggest a relation between the condition and cataleptoid states.

* 'Diagnosis of Diseases of the Spinal Cord,' 2nd edit., 1881, p. 29.

† If it should ultimately be proved (which is very improbable) that so short an interval as one fortieth of a second is sufficient for a reflex action, and that each contraction is reflex, the term "myotatic" will still be accurate, since it will remain true that tension is essential for the production of these contractions.

‡ By some interesting researches recently published, Mommsen has reached the same conclusion, that muscular tone is dependent on a muscle-reflex action excited by tension acting on the sensory muscle-nerves. ('Virchow's Archiv,' Bd. 110, p. 22).

§ 'Arch. f. Psych.,' Bd. x, p. 243.

CHANGES IN NUTRITION.—The nutrition of the tissue-elements is largely under the influence of the nervous system. Whether this influence is exerted through special “trophic” nerves, or merely through the motor, sensory, or especially through the vasomotor nerves, is a question that has been much discussed and is still undecided. Many disturbances of nutrition can be explained without the hypothesis of special trophic nerves. The fact regarding these changes that is best established and most important, is that the most acute disturbance of nutrition is the result of irritation of the nerve-trunks and centres, and is in proportion to the intensity of that irritation. If a thread is passed through each sciatic nerve, and one is also irritated by the application from time to time of irritant liquids, trophic changes occur in that limb with greater rapidity, and far greater intensity, than in the other. (Lewaschew.)

Nutritive changes in the skin are easily recognised, but they differ much in their character according to their acuteness. When they are rapid, and due to very acute irritation of the nerves or the spinal cord, such as attends acute myelitis, the temperature of the limb is raised, the vessels readily dilate, and remain dilated for a long time, bullæ form, containing a dark-coloured liquid, and slight pressure occasions a slough. These changes sometimes occur spontaneously, but are more often excited by some cutaneous irritation, and very slight irritation of the skin will suffice to produce them. Trifling pressure will set up a slough, and extensive vesication may result from the application of a hot-water bottle that is not more than pleasantly warm to a healthy hand. When the nerve irritation is intense, effusion may occur into the joints.

In the case of the fifth nerve, acute trophic changes occur in the eyeball chiefly when the disease involves the Gasserian ganglion, or the nerve in front of it. To produce the same effect, a lesion behind the ganglion has to be more irritating than one in front of it. We do not know whether a similar relation obtains in the case of the spinal nerves and the spinal ganglia. Very intense changes in nutrition are common in acute inflammation of the spinal cord.

The alterations in nutrition in chronic lesions (which have been carefully studied by Paget and Weir Mitchell) differ considerably from those that result from acute irritation. There is a slow change in the nutrition of the skin, which becomes red, thin, and shiny—the “glossy skin” of Paget. The subcutaneous tissue also wastes, so that the finger tips become pointed. The growth of the hair and nails is retarded, and the latter become brittle. The bones may suffer in their nutrition, and may break more easily than in health. If the lesion occurs during the period of growth, this is retarded, and the affected limb becomes shorter than the corresponding limb on the other side. We do not yet know by what nerve-fibres this influence on growth is exerted.

The *muscles* suffer in their nutrition from lesions of the motor nerves.

The bulk of a limb depends chiefly on the muscles, and therefore the interference with their nutrition is quickly shown by a diminution in the size of the part. At first there may be merely flabbiness, and an appearance of wasting that is not confirmed by measurement, but there is soon an actual diminution in the circumference of the limb. In extreme cases all the muscular tissue disappears, and the contour of the limb is considerably changed. In comparing the size of the limbs on the two sides, it should be remembered that the limbs on the right side are normally somewhat larger than those on the left side, and that the difference varies according to the occupation of the individual, and the extent to which this involves an excessive use of the limbs on one side. In measuring, great care is necessary to secure, as nearly as possible, the same conditions on each side, both as regards the state of the muscles and the place of measurement. It is best, wherever possible, to take the maximum measurement in each part, rather than to attempt to make the measurement in the same place. In the calf, the maximum circumference should always be taken. In the thigh, accurate measurement is extremely difficult, because a maximum cannot be taken. We may endeavour to measure at the same point in the thigh, at the same distance from the condyle, but it is very difficult to be exact, and a more accurate comparison can often be made by taking the minimum circumference above the knee, in spite of the fact that the muscular tissue there is small, and the difference less than it is elsewhere. In the forearm the best result is obtained by taking the maximum measurement around the muscular prominence below the elbow, over the supinator longus. In the upper arm, the circumference is nearly the same in the middle third, and the middle of the humerus may be measured, with little risk of error. In all cases care must be taken to draw the tape equally tight at each place. It is easier to do this with a flexible steel measure than with an ordinary tape measure. Exactness in this respect is best secured by having a spring at one extremity of the tape, with an index that will show the tension, which can thus be kept the same at each place.

ELECTRICAL IRRITABILITY.—The nerves and muscles are excitable by electricity, and the excitability is changed by disease, of which the change is often an important symptom. It indicates the state of nutrition of the nerve-fibres and muscles, and from this we can often draw important inferences regarding the condition of the centres. An outline of the most important facts regarding electricity in its application to medicine, is given in the Appendix, but it may be well to mention here the chief facts of diagnostic importance.

In the normal state, nerve-fibres are stimulated by either the induced or the voltaic current, the stimulation of the motor nerves being shown by contraction in the muscles supplied by them, that of the sensory nerves by the sensation that is caused. The contraction of the muscles is continuous when the faradaic current is applied, but if the

isolated shocks of which the current consists are separately passed, each causes a brief, momentary contraction. When the voltaic current is applied, contraction occurs, with a current of moderate strength, only when the strength of the current is changed, and chiefly when the current commences or ceases to pass, *i. e.* when the circuit is "made" or "broken." The stimulation of the sensory nerves is greatest at those times, but occurs also, in a much slighter degree, during the whole time that the voltaic current is passing. Hence this is probably the case also in the motor nerves, although the stimulation is too feeble to produce a contraction in health with a strength of current that can be borne. In proportion as the nutrition of the nerve-fibres is impaired, their excitability is lowered and a stronger current of each kind is required to excite them and cause contraction in the muscles they supply. When their nutrition is much impaired—*i. e.* when the fibres are "degenerated"—no contraction can be obtained even with the strongest currents.

The changes in the excitability of the muscles are less simple, because in them there are two excitable structures—the terminations of the nerves, and the muscular fibres themselves. Of these the nerve-fibres are the more sensitive to faradaism, and the faradaic stimulation of a muscle under normal circumstances is by means of these motor nerve-endings. Thus we find that its excitability corresponds in degree to that of the motor nerve supplying it. The muscular fibres themselves are, even in the normal state, less sensitive to faradaism than the nerve, apparently because they are incapable of ready response to a stimulus so very short in duration as are the shocks of which the faradaic current consists. (The proof of this consists in the fact that under the influence of curara, which removes the excitability of the terminations of the motor nerve, the muscle requires a stronger faradaic current to stimulate it than in the normal state.) But when the nerve is degenerated the slowly interrupted voltaic current stimulates the muscle as readily as in the normal state; a contraction occurs when the circuit is completed or broken—distinctly slower than that which occurs when the nerve-fibres are intact, and due to the stimulation of the protoplasm of the muscular fibres themselves. The fact that, under normal circumstances, the contraction which is caused by the voltaic current is as quick as that produced by the faradaic shock, is ground for believing that, in health, the voltaic, as well as the faradaic current, causes the muscle to contract chiefly by exciting the motor nerve-endings. When the motor nerve is degenerated, and will not respond to faradaic or voltaic stimulation, the muscle also loses all its power of response to the former. Apparently the nerve-degeneration is accompanied by changes in the nutrition of the muscular fibre, by which any power of response to faradaism, which it possessed in the normal state, is lost. But the response to the voltaic current remains, and becomes quickly more ready than in health, doubtless in consequence of nutritive changes which develope what

the older pathologists called, truly enough, "irritable weakness." Moreover, there may commonly be observed a change in the readiness of response to a certain mode of stimulation with voltaism—a "qualitative" change, as it is termed. In health, the first contraction to occur, on gradually increasing the strength of the current, is at the negative pole when the circuit is closed, and a stronger current is required before closure-contraction occurs at the positive pole. But, in the morbid state we are discussing, closure-contraction may occur at the positive pole as readily as at the negative, or even more readily—and contractions, when the circuit is broken, occur far more readily than in the normal state. This condition, then—faradaic irritability lost, voltaic irritability increased and often changed in quality—is termed the "degenerative reaction," because it occurs when the nerve-fibres are degenerated; and if we test *them* we shall find no response to any stimulus, voltaic or faradaic. It occurs when the nerves are separated partially or completely from their motor nerve-cells, and if no such separation exists, it indicates an acute degenerative change in those nerve-cells.

But the motor nerve-cells and fibres often undergo changes in nutrition of a much more chronic character. In this condition the irritability of the fibres is lessened gradually and slowly. The irritability of the intramuscular nerve-endings is lessened in the same degree as that of the nerve-trunks, and we have a diminution to both faradaism and voltaism. The nutrition of the muscular fibres is slowly, gradually, impaired; and when the nerve-fibres are much affected the muscular fibres are also. There is no stage in which the nerve-fibre irritability is lost, and the muscle-fibre irritability retained; hence there is no condition of lost faradaic and increased voltaic irritability such as characterises the degenerative reaction just described. Irritability is changed to the one form of stimulus just as to the other.

Between these two forms there are intermediate conditions. For instance, the nerves may present normal irritability, and the muscle the increased voltaic irritability and changed order of contraction met with in degeneration. Probably, in these cases, some nerve-fibres are degenerated, and lead to the increased irritability of some muscular fibres. In both nerve and muscle the character of the reaction is determined by the more irritable structures; hence it is normal in the nerve and altered in the muscle. This has been termed by Erb the "middle form" of degenerative reaction. It would be more accurate to call it the "mixed form."

The various changes in irritability have been thought to indicate the existence, and various affection, of separate centres for the nutrition of the nerves and muscles, apart from, though acting through, the motor nerve-cells. Remembering that the nerves and muscles contain fibres which suffer in different degrees, the phenomena at present ascertained may all be explained on the simpler principle stated, without

the assumption of these special centres, of the existence of which there is, indeed, no evidence.

Frequently the lowered irritability of degeneration in the nerves is preceded by a slight increase of irritability, very transient when the degeneration is acute, of longer duration when the degeneration is of the slower variety just noticed. In some morbid states, again, in which the change of nutrition in the cells and fibres is extremely slight, an increase may alone be discovered. I have found such an increase, for instance, in diseases regarded as functional, as paralysis agitans and chorea, and it is an interesting proof of the molecular changes which underlie, or result from, "functional" maladies.

THE MUSCLES: THEIR ACTION AND PARALYSIS.

Disease of the motor nervous system is largely manifested by loss of muscular action. Individual muscles, as well as groups of muscles, are often separately affected. Hence it is desirable to consider the symptoms of the paralysis of the more important muscles, before we enter on the study of special diseases.

The symptoms of the palsy of any muscle is a loss of its normal action, and a knowledge of this action is essential for the comprehension of those symptoms. The two must therefore be considered together. They are positive and negative aspects of the same facts. It may be well, at the same time, to mention the nerve by which each muscle is supplied.

The action of muscles is threefold. (1) By their tonic contraction they maintain the parts in a certain posture, independently of voluntary effort. By actual contraction they (2) produce certain movements, and also (3) oppose the action of other muscles by a feebler contraction and thus steady the movement that results.

The complex way in which muscles act together, and modify each other's effect, renders the subject a very large one. Here, only an outline can be given of the more important facts concerning the more important muscles. The reader who desires to pursue the subject further can do so in the 'Physiologie des Mouvements,' of Duchenne, to whose researches our present knowledge is largely due.

The DIAPHRAGM (phrenic nerves from the fourth and fifth cervical) although a double muscle with two nerves, habitually acts as a whole, the two halves contracting simultaneously and diminishing each lateral curve of the arch. The central tendon descends but little. The abdominal viscera are depressed, and the parietes protruded. If the hand is placed beneath the ribs, the descent of the viscera beneath the diaphragm can be felt. When the diaphragm contracts alone, as when the intercostals are paralysed, or the phrenic nerve is faradised,

the ribs to which the muscle is attached are slightly raised during its action, and this elevation causes a slight expansion of the thorax. In ordinary breathing this expansion is lost in the action of the intercostals. In paralysis the inspiratory protrusion of the upper part of the abdomen is lost; it even recedes during inspiration instead of advancing, and a descent of the viscera can no longer be felt by the hand. There often results a remarkable alternation in the respiratory movements of the thorax and abdomen, the retraction of the one corresponding to the protrusion of the other.

STERNO-MASTOID (spinal accessory nerve), passing from the sternum and adjacent part of the clavicle to the mastoid process, inclines the head towards, and rotates the face from, the side on which the muscle contracts. Both muscles together support the head in the vertical position, and, if it is bent back, they bring it forwards into, but not beyond, this position. Paralysis of one muscle has no influence on the position of the head, and but little on its movements. Other muscles supplement the loss. There is no such thing as a "paralytic torticollis." In palsy of both muscles, the head can be balanced in the vertical position, but if it falls back, it can be brought forward only with great difficulty. Each sterno-mastoid is associated in action with the muscles of the other side; it is a "contra-lateral muscle." For instance, in using the right arm, the head is turned to the right by the left sterno-mastoid. This association is sometimes reproduced in disease.

MUSCLES MOVING THE UPPER LIMBS.

MUSCLES MOVING THE SCAPULA AND SHOULDER-JOINT.—The *Trapezius* (spinal accessory, lower cervical and upper dorsal nerves) consists of three parts. The first, from the occipital bone to the outer end of the clavicle, is rarely used except in breathing (respiratory portion, Duchenne). The second part is that which passes from the lig. nuchæ, lowest cervical, and upper three dorsal spines, downwards and outwards to the acromion and outer part of the spine of the scapula. The lowest part passes from the dorsal spines below the third, outwards and partly upwards, to the inner half and base of the spine of the scapula. The second part is the chief elevator of the scapula and shoulder. With the third part it brings the scapula towards the spine, and puts the shoulder back. Both parts tend to rotate the scapula—acromion up, lower angle out. By this rotation the arm is carried above the horizontal level, to which the deltoid raises it. Paralysis of the highest part has little influence on the movement of the scapula, but causes a change in the contour of the neck (Fig. 5) especially conspicuous on deep inspiration. The change in the shape of the neck is very great, when the arms are raised, if the whole trapezius is wasted (Fig. 6). In palsy of the middle part, the elevation of the shoulder is imperfect; in that of the third part, the scapula is farther from the spine than normal. In palsy of all parts, the scapula becomes rotated (acromion down, inferior angle in) by the weight of the arm and the contraction of the opponents (Fig. 7). The rotation may mask the displacement outwards, due to the paralysis of the lowest part. If the clavicular part remains, there may be no rotation, but the scapula is lower than normal.

The *Rhomboids* (fifth cervical nerve, by a branch that passes through the scalenus) first rotate the scapula on the outer angle, moving the lower angle inwards, and then move the whole scapula upwards and inwards. In strong elevation, they aid the trapezius, which prevents the rotation of the scapula. The rotatory action aids forcible depression of the raised arm. The muscles also

fix the scapula for the action of the *teres major*. Their tone helps to keep the scapula against the thorax (opposing the *pectoralis*) and in its vertical



FIG. 5.—Paralysis and wasting of trapezius; alteration in contour of shoulder at rest.



FIG. 6.—Ditto, when the arms are raised (the right one being aided by another person).

position (opposing the *serratus*), and hence, in paralysis, the edge of the scapula, at rest, stands out a little, leaving a furrow, and the scapula is slightly rotated (lower angle out). Movement is but little interfered with by the paralysis of the rhomboids, the most important effect being that the movement backwards of the raised arm by the *teres* and *deltoid* is feeble for want of the fixation of the scapula.



FIG. 7.—Paralysis and wasting of deltoid and trapezius; rotation of scapula, from weight of arm, in consequence of the paralysis of the trapezius; progressive muscular atrophy.

The *Levator anguli scapulae* (third cervical nerve) first rotates the scapula on the outer angle and then raises it. The muscle is usually paralysed with the trapezius, and then the scapula falls, but the special effect of its palsy is lost in that of the trapezius. If the levator is preserved, and the trapezius paralysed, there is great rotation of the scapula, which is, as it were, suspended by its inner angle (Fig. 7).

Serratus magnus (posterior thoracic nerve, from the fifth and sixth cervical) carries the scapula outwards, forwards, and slightly upwards, when the arm is put forwards. It tends to rotate the scapula on the inner angle (acromion up), the lower fibres most powerfully, but this rotation is prevented by the rhomboids and levator anguli. It does not raise the shoulder when the arm is hanging. It helps to fix the scapula when the posterior fibres of the deltoid move the raised arm back. If the scapula is fixed by the rhomboids, the *serratus* can act on the ribs, and aid forced inspiration. It has most inspiratory effect when the arms are elevated. In paralysis there may be little change in the position of the scapula at rest, but often there is slight rotation (lower angle in) from the unopposed tone of the rhomboids. When the arm is moved forwards by the anterior part of the deltoid, the scapula, no longer held against the thorax and

moved forwards by the serratus, is rotated on its vertical axis by the action of the anterior part of the deltoid on the humerus, and of the middle part on the scapula. Thus the posterior edge recedes from the thorax, leaving a groove into which the hand can sometimes be placed (Fig. 8). The scapula is, at the same time, rotated; lower angle inwards and upwards. Elevation of the arm above the level of the shoulder is much weakened, but can be imperfectly effected by the middle part of the trapezius. Loss of the serratus weakens other movements, but does not abolish any. Inspiratory expansion of the thorax, when the arms are raised, is distinctly less on the paralysed side (Poore).

The *Deltoid* (circumflex nerve, from the four lowest cervical) abducts the humerus, the anterior and posterior fibres also moving the arm forwards and backwards respectively. The arm is raised least by the posterior, and most by the anterior fibres, but even the latter

only elevate it to a right angle with the trunk. Hence, if raised by the anterior fibres, and then moved back by the posterior, it is at the same time depressed. Elevation above a right angle is by rotation of the scapula (trapezius and serratus). These muscles also fix the scapula for the deltoid, preventing the rotation (acromion down, lower angle in) that the deltoid, acting alone, would cause. In paralysis, abduction of the arm, direct, forwards, and backwards, is almost lost. All the abduction that remains is a trifling movement by the supraspinatus. An attempt to abduct results in rotation of the scapula and elevation of the shoulder (Fig. 9) from an excessive innervation of the associated

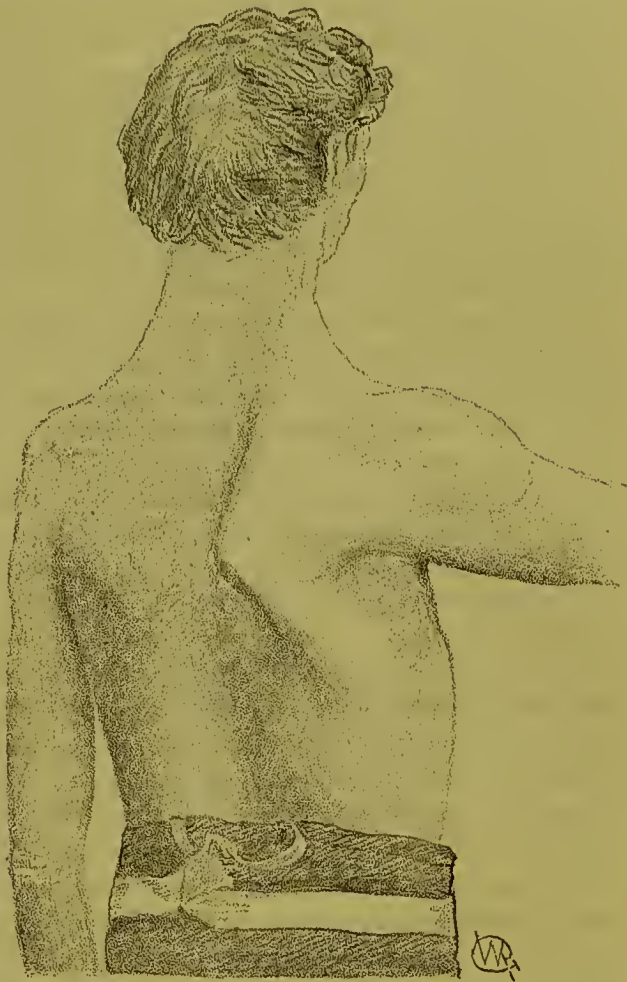


FIG. 8.—Paralysis of the serratus magnus; eversion and rotation of scapula when the arm is put forwards.



FIG. 9.—Paralysis of right deltoid; elevation of shoulder by trapezius on an attempt to raise the arm, which is slightly abducted by the supraspinatus.

trapezius and serratus, which, as we have seen, fix the scapula when the deltoid acts. Paralysis of single parts of the deltoid causes loss of the corresponding movement of the arm, but if the middle part only is paralysed, there is still a limited power of direct abduction by the conjoined contraction of the anterior and posterior parts, aided by the supraspinatus.

The *Supraspinatus* (suprascapular nerve) abducts the arm, moves it forwards, and rotates it in. It thus aids the deltoid. Isolated paralysis of the supraspinatus has little influence on movement or position, but if the deltoid is also paralysed, the head of the humerus falls away from the acromion far more than when the deltoid is paralysed alone.

The *Infraspinatus* (suprascapular nerve) rotates the humerus outwards, and in paralysis this movement is lost. A difficulty in writing is produced, the movement along the line being by this rotation of the humerus.

The *Teres minor* (circumflex nerve) has a similar action to the infraspinatus, and its palsy has a similar effect.

The *Subscapularis* (short subscapular nerve, from the fifth and sixth cervical) rotates the humerus in, and its paralysis lessens this movement.

The *Latissimus dorsi* (long subscapular nerve, from the brachial plexus—last four cervical nerves) lowers the raised arm, and puts it back; the upper part adducts the scapula, the lower depresses the shoulder by acting on the humerus, which it tends to drag out of the socket. It inclines the trunk a little, and both muscles together extend the trunk. In paralysis, forcible backward depression of the raised arm is lost, and the shoulder cannot be put back without being also raised (by the trapezius).

The *Pectoralis major* (anterior thoracic nerves from the brachial plexus) consists of two muscles, the action of the clavicular and sternal parts being different. The clavicular (which arises also from the highest part of the sternum), if the arm is hanging, brings the shoulder forwards and upwards, as in shivering; if the arm is raised, it is brought forwards and lowered to the horizontal position. The muscle is thus concerned, Duchenne says, in the "cut" of the swordsman and the benediction of the priest. The sternal portion lowers the raised arm from every position, and if the arm is hanging, it draws the shoulder down. Paralysis of the upper part has little effect on the movement of the arm, because the anterior fibres of the deltoid have the same action. It is easily recognised by making the patient put his arms in front of him and press the palms together. In paralysis of the lower part, even with the latissimus, the raised arm can still be lowered accurately, by the weight of the arm and relaxation of the elevators, but it cannot be lowered against even a slight resistance. Thus a blacksmith with this defect could wield a heavy hammer, but could not blow the bellows by pulling downwards a cord (Duchenne).

The *Teres major* (short subscapular nerve) approximates the humerus and the outer edge of the scapula, by bringing the former to the side of the trunk, and rotating the latter. The simultaneous contraction of the levator anguli and rhomboids, fixing the inner angle, causes this rotation to raise the prominence of the shoulder. Hence, in a forcible elevation of the shoulder, the arm is pressed against the side, the lower fibres of the latissimus and pectoralis major aiding the adduction. The teres cannot alone put the arm behind the trunk. In paralysis, the elevation of the shoulder, with the arm against the side, is lost.

MUSCLES MOVING THE FOREARM.—*Triceps* (musculo-spiral nerve).—The long head has an action similar to the teres, but feebler. It contracts when the arm is forcibly lowered, and prevents the displacement downwards of the head of

the humerus by the actual depressors, the latissimus and pectoralis. All parts extend the elbow, the long head with less force than the others, but its action on the shoulder-joint, just mentioned, is important, because forcible depression of the raised arm is often associated with extension of the elbow. In paralysis of the triceps, the elbow can only be extended by the weight of the forearm, and extension against gravitation is impossible. Thus a man with paralysis of the triceps cannot raise his hat in the customary manner. Flexion of the elbow is uncertain, on account of the loss of the antagonistic steadying force.

The *Brachialis anticus* (musculo-cutaneous and musculo-spiral nerves) flexes the elbow simply. Its rare isolated palsy has little effect, since it is supplemented by the biceps and supinator longus.

The *Biceps* (musculo-cutaneous nerve) supinates the forearm if it is pronated, and then flexes the elbow. In paralysis the flexion can still be effected, but the traction on the humerus causes pain at the shoulder, from the loss of the support of the long head of the biceps.

The *Supinator longus* (musculo-spiral nerve) places the forearm midway between pronation and supination, and then flexes the elbow. If it is paralysed, there is a tendency for supination to accompany flexion (biceps), and if the brachialis anticus is also paralysed, the elbow can only be flexed when the forearm is supinated.

If the three direct flexors of the elbow are paralysed, feeble flexion is still possible by the extensors of the wrist, which cross the elbow-joint, but only after the forearm has been pronated, and the wrist over-extended.

Pronators.—The *p. teres* and *p. quadratus* (median nerve) both pronate strongly, and their palsy causes loss of this movement, but pronation to the mid-position is still possible by the supinator longus.

The *Supinator brevis* (musculo-spiral nerve by posterior interosseal branch) is the only simple supinator. If it is paralysed, supination can still be effected by the biceps, and also by the supinator longus as far as midway between pronation and supination.

MUSCLES MOVING THE HAND.—*Flexors of Wrist.*—The *f. carpi ulnaris* (ulnar nerve) and *f. carpi radialis* (median) flex the wrist. The ulnar tends to turn the supinated hand still more out, but they do not move the wrist-joint laterally. Flexion indeed hinders the lateral movements of the wrist, in consequence of the shape of the articular surfaces. The *f. c. ulnaris* flexes the fifth metacarpal bone on the carpus, as well as the wrist-joint. In paralysis, flexion of the wrist can only be effected by the flexors of the fingers when these are extended. Extension of the wrist is unsteady from the loss of the synergic contraction of the flexors.

Extensors of Wrist.—*E. carpi radialis brevis et longus: e. c. ulnaris* (musculo-spiral nerve and its radial branch). The short radial is a direct extensor; the long radial and the ulnar move the hand laterally as well. In paralysis of all three extensors, the wrist can only be extended by the extensors of the fingers when the phalanges are flexed. In loss of the short radial, direct extension is still possible by the long radial and ulnar, and lateral extension by one of these alone. If either is paralysed, together with the short radial, direct extension is lost, and only lateral extension, in the direction of the remaining muscle, is possible. Paralysis of either lateral extensor, long radial or ulnar, leads to permanent deviation of the wrist in the direction of the remaining muscle (Fig. 10). The loss of the long radial is more serious than that of the ulnar, because the radial lateral movement is of more importance, being needed for the convenient movement of the hand to the mouth. Paralysis of the extensors

impairs flexion of the fingers, from the great shortening of the course of the tendons, by the flexion of the wrist that occurs. The ulnar extensor acts also synergically with the extensor of the metacarpal bone of the thumb, as may be noted if the finger is placed on the tendon beneath the styloid process of the ulna. Hence, in paralysis of this extensor, the hand deviates laterally when the thumb is strongly extended.



FIG. 10.—Paralysis of the long radial extensor of the wrist in a young child; habitual deviation of the hand towards the ulnar side. (After Duchenne.)

Extensors of the fingers; Extensor communis digitorum; E. indicis; E. minimi digiti (musculo-spiral nerve). The common extensor moves the fingers and then the wrist. When the muscle is faradised, the extension begins at the distal phalanges, and these become flexed again, when the hand is extended beyond the plane of the forearm, by the tonic force of the flexors, the course of their tendons being elongated by the extension of the wrist. But physiologically the muscle has little action on the last two phalanges, since they cannot be extended by the long extensor, if the interossei, their proper extensors, are paralysed. During extension by the communis the fingers are separated from the second. The extensors



FIG. 11.—Posture of the hand in contraction of the palmar fascia, resembling that in paralysis of the long extensors of the finger. (From a sketch by Mr. V. Horsley.)

of the first and last fingers have a similar extensor action, but, in addition, they adduct their respective fingers towards the middle finger. In paralysis the extension of the fingers is impossible, but if the proximal phalanges are passively extended, the middle and distal joints can be extended by the interossei. For the lateral movements of the digits, extension of the proximal phalanges is essential, and hence these movements are lost, but they can be performed if the proximal phalanges are passively extended. The posture of the fingers due to contraction of the palmar fascia resembles that in palsy of the long extensor (see Fig. 11), but an examination of the palm shows the cause of the flexion.

Flexors of fingers; F. sublimis (median nerve); *F. profundus* (median and ulnar nerves). These muscles flex chiefly the second and third phalanges, the first phalanx being flexed by the interossei. The superficial muscle flexes the second phalanx on the first, the deep flexes both. The action on the first phalanx is confined to extreme flexion of the fingers, and is the less the more the wrist is flexed. But if the flexion of the middle and distal phalanges is prevented, the first is strongly flexed. In extreme shortening of the course of the tendons by flexion of the wrist, the action on the fingers is very feeble, and hence there is a normal synergic action of the extensors of the wrist. When the extensor of the fingers is in strong action, extending the proximal phalanges, the action of the flexors on the second and third joints is very strong (tearing position). In paralysis of these muscles the power of flexing the last two joints is lost, but the interossei still flex the metacarpo-phalangeal joints. Paralysis of the deep

flexor alone causes loss of the power of flexing the distal joint, but interferes with many movements, such as playing on the piano. In paralysis of these muscles, the unopposed tone of their opponents, the interossei, which extend these joints, leads in time to over-extension, and, with repeated passive pressure in using the fingers, may even produce a subluxation backwards. In palsy of the sublimis this effect is chiefly seen at the middle joint (Fig. 12), in that of the profundus as the distal joint.

Interossei and lumbricales (ulnar nerve, except the outer two lumbricales, which are supplied by the median). The Interossei abduct and adduct the fingers, but only when these are extended at the metacarpo-phalangeal joints, and some effort is required for adduction, since the tendency of the long extensor is to separate the fingers, and this influence has to be overcome. They also extend the second and third phalanges on the first, and flex the first on the metacarpal bones. The lumbricales aid the flexor-extensor action of the interossei, but do not move the fingers laterally. The opposite action of the forearm muscles and of the interosseal extensors and flexors is very important. Their synergic action steadies movements, and in many actions they contract alternately. Thus in making a down-stroke with a pen or pencil, the long flexors bend the last two joints; while in making an upstroke, these are extended, and the metacarpo-phalangeal joint is flexed, by the interossei. In paralysis of these muscles the lateral movements are lost, but a slight abduction

and adduction of the index can still be effected by its long extensors. Only the first phalanx can be extended, and flexion is almost confined to the last two phalanges. The first three lumbricales, being supplied by the median nerve, often escape when the other muscles are paralysed by an injury to the ulnar nerve, and these aid the others when they are merely weak; hence the index and middle fingers seem to recover before the others (Fig. 13). The position of the hand at rest becomes altered. Normally there is slight flexion at all joints



FIG. 12.—Paralysis of the fibres of the flexor sublimis which act on the two middle fingers: twelve years' duration. The second phalanges of these fingers are bent backwards and subluxated from the contracture of the unopposed interossei, while the last phalanges are kept in position by the unaffected flexor profundus. (After Duchenne.)

FIG. 13.



FIG. 14.

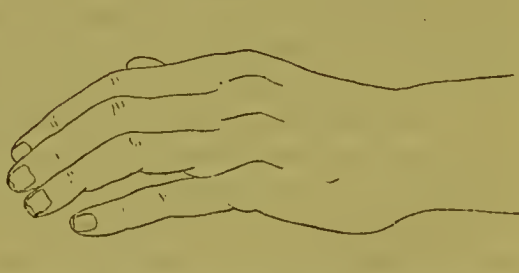


FIG. 13.—Recent incomplete paralysis of the interossei from a punctured wound of the ulnar nerve at the wrist: attempt to extend fingers. The loss of extension of the last two phalanges is chiefly marked in the last two fingers, from the influence of the lumbricales (supplied by the median) on the others. (After Duchenne.)

FIG. 14.—Paralysis of the interossei (ulnar nerve) slight in degree: attitude of fingers at rest.

by the tone of the muscles, interossei and long flexors. In paralysis the first phalanx is in a line with the metacarpal bones, while the other phalanges are flexed, the middle more than the distal (Fig. 14). In action this flexion is always increased, the metacarpo-phalangeal joints become over-extended, and the other joints strongly flexed (Fig. 15). Gradually the hand assumes this

FIG. 15.



FIG. 16.



FIG. 17.



FIG. 18.



FIG. 15.—Attempt to unbutton waistcoat by the hand shown in the last figure; extreme flexion of the two last phalanges, and extension of the first, on the attempt to use the fingers. (From nature.)

FIG. 16.—Old-standing palsy of interossei and thenar muscles, showing the over-extension of the first and flexion of the two last phalanges.

FIG. 17.—Paralysis of all the intrinsic muscles of the hand and of the long flexor of the thumb, in consequence of an injury to the brachial plexus in dislocation of the shoulder. The fingers present the claw-like attitude, the thumb is in extension. (After Duchenne.)

FIG. 18.—Paralysis of the ulnar nerve from a wound at the wrist (indicated in the figure). Extreme claw-like hand from the unopposed contraction of the common extensor and long flexors of the fingers and thumb. (After Duchenne.)

posture even at rest (Fig. 16), and ultimately the posture becomes warped into a deformity by the over-extension of the first phalanges, and extreme flexion of the others, due to the contracture of the long extensor and of the flexors; the tendons of these muscles stand out conspicuously on the back and in the palm, and a claw-like attitude is developed, the "main en griffe" (Figs. 17 and 18). Changes in the articulations may ultimately limit even passive movement.

MUSCLES OF THE THUMB.—*Extensor secundi internodii pollicis* (musculo-spiral nerve) extends both phalanges, and moves the whole thumb backwards and from the fingers, so as to bring it behind the plane of the metacarpus. It may ultimately extend the wrist-joint, but it never supinates. It is not used in extending the thumb when this is opposed to the first finger. In paralysis, the metacarpal bone of the thumb is slightly flexed on the carpus, and is inclined forwards. The second phalanx is flexed on the first, and can only be extended (by the abductor and outer part of the short flexor) when the metacarpal bone is adducted, and the first phalanx is flexed. The constant flexion of the second phalanx interferes with the movement of the index finger, unless the

patient remembers to move the thumb out of the way by the extensor of the metacarpal bone. Writing is not interfered with because the muscle is not concerned in extension with opposition.

The *Extensor primi internodii pollicis* (musculo-spiral nerve) is the true abductor of the thumb. It moves the metacarpal bone outwards, and extends the first phalanx. It would move the whole hand in the same direction as the thumb, were not this tendency counteracted by the synergic contraction of the extensor carpi ulnaris (q. v.). It does not pronate or supinate. In paralysis, abduction of the metacarpal bone is less than normal. There is an undue flexion of the first phalanx, and the metacarpal bone is flexed on the carpus, so that the thumb is drawn towards the palm. The loss of this muscle is, however, compensated to a considerable extent by other muscles.

The *Extensor ossis metacarpi pollicis* (musculo-spiral nerve) is really the long abductor of the thumb. It moves the metacarpal bone outwards and forwards, flexing it on the carpus, and then flexes the wrist with slight pronation. It thus moves the thumb as much forwards as outwards. In *paralysis* the metacarpal bone is, at rest, less inclined forwards than normal, and somewhat abducted, but the first phalanx is in its normal position. Movement of the thumb is but little interfered with.

In combined palsy of the extensors of the first phalanx and of the metacarpal bone (common in lead palsy), the thumb becomes adducted, and is parallel to the radius. The first phalanx is slightly flexed by the thenar muscles.

The *Thenar muscles* constitute two groups: (1) The short *abductor and outer portion of the short flexor* (median nerve) move the metacarpal bone forwards and inwards (flexing the first phalanx), incline it outwards, and rotate it inwards, so as to place its palmar aspect opposite the fingers. The second phalanx is ultimately extended. If the metacarpal bone is previously abducted the movement is greater and amounts to circumduction. (2) The *adductor and inner part of short flexor* (ulnar nerve) go to the inner side of the first phalanx. The metacarpal bone is moved towards that of the second finger; if previously flexed, it is extended; if previously opposed to the index, it is moved a little outwards. The phalanges follow the movements of the metacarpal bone, but the first is slightly flexed and the second is extended, as the fingers are by the interossei.

The *Opponens pollicis* (median nerve) flexes the metacarpal bone on the carpus, and abducts it, but this movement is insufficient to oppose the thumb to the index; the conjoint action of the abductor is necessary (see above).

The *Flexor longus pollicis* (median nerve) flexes the second phalanx forcibly and the first feebly. It has no action on the metacarpal bone. It is used in writing (making a stroke towards the body) and in picking up a small object, &c. In paralysis this flexion is lost, and with it these actions; if an object is held between the tips of the thumb and forefinger, the last phalanx of the thumb is bent back. Other movements of the thumb are not interfered with.

If all the thenar muscles are paralysed, the metacarpal bone is in the plane of the index, and drawn towards it by the extensor of the second phalanx, which moves the metacarpal bone inwards and backwards. The whole thumb corresponds with the metacarpal bone in position, the phalanges being normal. In paralysis of the short abductor and flexor, the second phalanx cannot be extended unless the metacarpal bone is abducted. These short muscles normally prevent the abduction that the extensor of the second phalanx tends to produce. The latter, moreover, prevents undue adduction when the special abductors are paralysed, but brings the metacarpal bone into the plane of the index. Thus the

posture of the hand at rest resembles that of the hand of an ape (Figs. 19 and 20). If the abductor and opponens are paralysed, the tips of the thumb and fingers can

FIG. 19.

FIG. 20.

FIG. 21.



FIG. 19.—Normal position of the thumb (for comparison with the succeeding figures.)

FIG. 20.—Position of the hand in long-standing paralysis and wasting of the thenar muscles. Under the influence of the long extensor the metacarpal bone of the thumb has been brought into the same position as the other metacarpal bones, being rotated slightly, so that the back of the thumb is in the plane of the back of the hand, like the hand of the ape. (After Duchenne.)

FIG. 21, from another case, shows the same condition, but still greater displacement of the metacarpal bone has taken place, from the greater contraction of the extensor. (After Duchenne.)



FIG. 22.—Paralysis of the abductor brevis and opponens pollicis. From the want of these muscles the thumb can only be brought in contact with the tip of the index by strong flexion of the last two phalanges of the fingers, otherwise the tip of the thumb only reaches the middle of the second phalanx. (After Duchenne.)

only be brought together by flexing the last phalanges of the digits (Fig. 22). Then the thumb can be brought into contact with the finger by means of the short flexor, which inclines the metacarpal bone sufficiently to effect this, although not enough for the tip of the first finger to touch the thumb when its phalanges are extended.

If the short flexor is paralysed, the thumb can still be opposed to the first two fingers by the abductor, but it cannot be opposed to the two last fingers, on account of the deficient lateral inclination of the thumb, which should be produced by this muscle. Writing is easy by means of the short abductor, whereas, if this is lost, although the thumb can be opposed to each of the fingers, writing is much interfered with.

If all the thenar muscles are paralysed, a certain amount of opposition of the thumb and fingers is still possible, by means of the flexion of the last phalanges of the thumb and fingers. If all are paralysed except the adductor, objects can still be held between the thumb and side of the palm.

MUSCLES OF THE LOWER LIMB.

MUSCLES MOVING THE HIP-JOINT.—The *Gluteus maximus* (small sciatic nerve chiefly) extends the hip-joint, and feebly rotates the thigh outwards. It is the most powerful extensor of the hip, and it is chiefly used when a forcible

extension is required, and the joint has been previously flexed. It is employed, not in standing, or in walking on level ground, but in going upstairs, or uphill, and in rising from a seat. When it is paralysed these movements are difficult.

The *Gluteus medius* (gluteal nerve) is the chief abductor. All parts of the muscle have this action, but, in addition, the anterior third moves the thigh forwards and rotates it inwards, while the posterior third moves it backwards and rotates outwards. The successive action of the several parts causes circumduction. The *Gluteus minimus* (gluteal nerve) has probably the same action. In paralysis, abduction and circumduction are lost; in standing on the other foot the pelvis is inclined from the affected side, and hence, in walking, there is an oscillation of the trunk, which becomes very considerable if the muscles of both sides are affected. Moreover, the unopposed tone of the outward rotators produces a permanent rotation of the leg, so that the toes are directed outwards, and, from the altered position of the foot, the propulsion of the body in walking is deficient.

The *Pyriformis*, *Gemelli*, *Obturator internus*, and *Quadratus femoris* (special nerves from the sacral plexus), all rotate the thigh outwards, and the first-named muscle, in addition, carries the thigh obliquely backwards and outwards, in the same way as the posterior fibres of the *gluteus medius*. In paralysis of these muscles, external rotation is impossible, and the unopposed tone of the internal rotators (anterior fibres of the *gluteus medius* and *minimus*) causes the leg and foot to be habitually turned inwards.

The *Psoas* (lumbar nerves) and *Iliacus* (anterior crural nerve) flex the hip-joint, and, in doing so, cause also a slight rotation outwards. In paralysis flexion is lost, and the use of the leg in walking becomes impossible.

The *Tensor of the Fascia lata* has a slight power of flexing the hip, and at the same time rotates the thigh in. It normally counteracts the tendency of the ilio-psoas to rotate outwards. If it is paralysed, there is a tendency for the foot to turn out when it is being brought forward in the act of walking.

Adductors of the Thigh.—The *Pectineus* (obturator nerve) causes an oblique movement forwards and inwards, *i.e.* a combined flexion and adduction, as in crossing the legs. It also rotates outwards. The *Adductor longus*, and probably the *Adductor brevis* (obturator nerves), have the same action, but the flexion is less than by the pectineus. The *Adductor magnus* (obturator and great sciatic) causes a similar adduction, but while its upper fibres rotate outwards, its lower fibres rotate in, and are employed in keeping the foot straight during adduction in riding. This is very difficult, if these fibres are paralysed. The foot then turns out when the hip is flexed, either in the recumbent posture, or in walking, from the preponderance of rotation out by the other adductors. When all the adductors are paralysed, not only is adduction lost, but in flexion of the hip the foot is moved forwards and outwards, instead of directly forwards, showing that there is normally a synergic action of the abductors and adductors with the flexors in this movement.

MUSCLES MOVING THE KNEE.—*Extensors of the Knee: Rectus and Vasti* (anterior crural nerve).—The vasti act solely on the knee-joint; the rectus also aids in flexing the hip, but chiefly when the knee is bent. In consequence of its passage over the hip-joint, moreover, the force with which it extends the knee is increased by the simultaneous extension of the hip. This effect is useful in the propulsion forwards of the body in walking.

In paralysis of the extensors of the knee, standing is still possible if the knee is extended, since the arrangement of the articulation renders a contraction of

the extensors unnecessary. But secondary shortening of the flexors is apt to occur, and then standing becomes impossible because the knee cannot be perfectly extended. In the same way, walking is possible if the leg is not moved forward beyond the vertical position; if it is, the knee becomes flexed by the weight of the leg and foot, and the patient falls when he attempts to rest upon it. Rising from the kneeling posture in the ordinary way is impossible. In partial paralysis of the muscles, as in pseudo-hypertrophic paralysis (q. v.), the extension of the knee, in rising, is facilitated by placing the hand upon it, and so bringing the centre of gravity of the body near the fulcrum of the lever formed by the femur. If the vastus internus and rectus are paralysed, the vastus externus may dislocate the patella by the obliquity of its traction. The vastus internus never does so in the opposite condition because its action is less oblique.

Flexors of the Knee.—The *Sartorius* (ant. erural nerve) flexes the hip and knee-joints, and has a feeble power of rotating the thigh outwards and the knee inwards. It is a muscle of small importance.

The *Gracilis* (obturator nerve) adducts the thigh more powerfully than it flexes the knee. It rotates the leg inwards.

The *Semitendinosus*, *Biceps*, and *Semimembranosus* (great sciatic nerve) are not only flexors of the knee but extensors of the hip-joint, and are the muscles that extend the hip during ordinary walking, the gluteus maximus (q. v.) being called into action only during special efforts. The leg is rotated inwards by the semitendinosus, outwards by the biceps.

In paralysis of the flexors the resulting loss of the power of flexion interferes with walking, since the knee-joint cannot be bent, in the forward movement of the leg, until the thigh is flexed sufficiently to permit the weight of the foot to flex the knee. To prevent the toes striking the ground the foot is unduly flexed on the leg. The loss of the support that the flexor tendons give to the knee-joint leads to an undue strain on the ligaments, which become stretched, and slight retroflexion of the joint may occur.

In paralysis of the muscles that extend the hip there is a tendency to fall forwards in walking. To counteract this the trunk is carried backwards, and a fatiguing strain on the flexors of the hip results.

In paralysis of the biceps, the leg, during flexion, is rotated inwards; when the biceps remains and the other muscles are paralysed, there is an undue rotation outwards. The effect of these abnormal movements on the ligaments of the joint is such that, after a time, the amount of rotation becomes greater than is possible in health.

The *Popliteus* (internal popliteal nerve) has but a feeble power of flexing the knee. Its chief action is to rotate the leg inwards when the knee-joint has been flexed.

MUSCLES MOVING THE FOOT.—*Extensors of foot on leg.** The *Gastrocnemius* and *Soleus* (internal popliteal nerve, from the sciatic) have the same action. They extend the hinder part of the foot and draw down the outer side of the fore part of the foot, but very little the inner side. Hence the foot is rotated, so that the dorsum looks outwards, while the whole foot is turned inwards on the axis of the leg. The peculiar inversion and adduction that thus accompanies extension is due to the form of the articular surfaces. The gastrocnemius has very little power of flexing the knee, but the extension of the knee increases the

* These muscles have lately been termed "plantar flexors," because they are homologous with the flexors of the wrist. The term is a bad one, since it involves a use of the word "flexor" in absolute contradiction to its proper signification.

effect of the muscle on the ankle-joint, especially in walking, just as we have seen that the extension of the hip augments the force with which the contracting rectus extends the knee. For direct extension of the ankle, the peroneus concurs and opposes the inversion. The only difference between the gastrocnemius and soleus is that the latter, having no attachment to the femur, can extend the ankle when the knee is flexed as well as when it is extended. In paralysis of these muscles extension of the ankle (by the peroneus longus and flexor longus digitorum) is extremely feeble, and the foot can scarcely be carried beyond a right angle. Walking is greatly interfered with; standing on tiptoe is impossible. The unopposed peroneus longus causes eversion of the foot, lowers the head of the first metatarsal bone, and deepens the plantar arch. In time the ankle-joint becomes over-flexed; the heel considerably lowered and the plantar muscles and fascia become shortened. The resulting deformity is termed talipes calcaneus (Fig. 23).



FIG. 23.—Talipes calcaneus from atrophic paralysis of the calf muscles, with flexion of the middle and distal phalanges of the toes from paralysis of the interossei.

Peroneus longus (museulo-cutaneous nerve, from external popliteal of sciatic) everts the foot, lowering the inner border, narrowing the foot, and increasing the plantar arch. It also turns the whole foot out on the axis of the leg. It has a very feeble power of extending the ankle. It keeps down the inner part of the foot during extension by the calf muscles, as in walking. In paralysis of this muscle the inner part of the front foot is not supported during extension, and yields to slight force. The foot becomes adducted and rotated, so that the sole is directed inwards, in consequence of the unopposed action of the sural muscles. The inability to press the inner part of the ball of the foot firmly against the ground leads to over-action of the flexors of the great toe. The plantar arch is lessened; there is "flat-foot."

Flexors of the Foot.—The *Tibialis anticus* (anterior tibial branch of ext. popliteal nerve) produces simultaneously three movements: it elevates the inner part of the front-foot (opposing the peroneus longus); it flexes the ankle-joint, and adducts the foot. The *Extensor longus digitorum* (ant. tibial nerve), besides extending the toes, flexes and abducts the foot. The abduction is in consequence of the outward position of its tendons beneath the annular ligament. These two muscles together produce direct flexion of the foot, or flexion with adduction or abduction, as the force of one or the other preponderates. Paralysis of either weakens flexion, and the corresponding lateral movement is lost, flexion being accompanied by the deviation effected by the muscle that remains. The defect in flexion is greatest in paralysis of the tibialis, and the loss of the instinctive flexion, when the leg is brought forward in walking, causes the foot to catch

against the ground. Paralysis of the flexors is followed by secondary contracture of the extensors, and talipes equinus results (Fig. 24), which is the greater



FIG. 24.—Talipes equinus, due to atrophy of the tibialis anticus and secondary contracture of the calf muscles. In A the foot is shown at rest; there is slight equino-varus. In B it is shown during flexion, and the varus is changed to valgus by the action of the peroneus longus. Note the increased extension of the toes in B from the compensatory over-action of the long extensors of the toes. (After Duchenne.)



FIG. 25.—Extremetalipes equinus from old-standing palsy of the tibialis anticus (infantile paralysis) and extreme contraction of the calf muscles. No flexor movement was possible.

the longer the palsy has lasted (Fig. 25). Its occurrence is facilitated, in many cases of palsy, by lessened growth of the bones of the leg, so that the ball of the foot only touches the ground when the foot is extended. There is usually slight rotation inwards of the foot at rest, even when the tibialis is paralysed (see Fig. 24, A), because such rotation is produced by the sural extensors (p. 34), but in this case the slight valgus at rest is changed to varus on an attempt to flex the ankle (Fig. 24, B).

The *Peroneus brevis* (musculo-cutaneous branch of ext. popliteal nerve) abducts the foot and rotates it, raising the outer edge.

The *Tibialis posticus* (posterior tibial nerve from int. pop.) adducts the foot and curves it, rendering the outer border and instep more convex. Its power of adduction is greater than that of the tibialis anticus, and it does not rotate the foot in the same manner.

These two muscles alone have the power of adducting and abducting without flexing or extending; and in their paralysis these simple movements are lost. If one only is paralysed, a deformity develops corresponding to the action of the other muscle,—talipes valgus in paralysis of the tibialis posticus; t. varus in that of the peroneus brevis.

The *muscles moving the toes* present, in their mode of action, a close correspondence to those of the fingers.

The *Extensor longus digitorum* and the *Extensor longus pollicis* (anterior tibial nerve) extend chiefly the first phalanges, while the *Flexor longus digitorum* and *Flexor brevis* (posterior tibial nerve) flex the last two phalanges. The

Lumbricales and the *Interossei* (post. tibial nerve by ext. and int. plantar), together with the *Abductor* and *Flexor brevis minimi digiti*, oppose both the other extensor and flexor muscles, flexing the first phalanx and extending the others. This action is of great importance in walking, since they give the last propulsion to the body as the ball of the foot leaves the ground. The *Abductor*, *Adductor*, and *Flexor brevis pollicis* (plantar nerves from post. tibial), have a similar action on the great toe, but with adduction or abduction respectively. The *interossei* also produce a lateral movement of the toes, but this action is of little practical importance. In paralysis of the common extensor of the toes, and of the proper extensor of the great toe, the tonic force of the *interossei* and analogous muscles produces persistent flexion of the first phalanges and extension of the others. If the conditions are reversed, and the latter muscles are paralysed, the first phalanges are over-extended, sometimes even subluxated, and the two other joints are flexed, so that a claw-like form of foot is the result (Figs. 26 and 23). The final propulsion in walking, above described, is much interfered with, and the attempt is painful because the ends of the toes are turned towards the ground.



FIG. 26.—Paralysis of the *interossei* and the adductor and short flexor of the great toe. The first phalanges are over-extended and the second are flexed, while the hollow of the sole is increased. (After Duchenne.)

PART II.

DISEASES OF THE NERVES.

GENERAL PATHOLOGY.

STRUCTURE.—The individual fibres consist of a central “axis-cylinder,” which is the chief functional element, surrounded by the

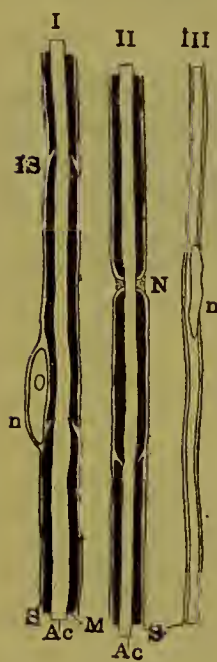


FIG. 27.—Diagram of the structure of nerve-fibres. I and II, medullated, III, non-medullated fibre; S, sheath; M, myelin, or white substance of Schwann; Ac, axis cylinder; n, nucleus; N, node; IS, incision of Schmidt. (From a preparation of the nerve-fibre of a frog, stained with osmic acid.)

“medullary sheath,” or “white substance of Schwann,” composed of myelin (M, Fig. 27). The latter is absent in the “non-medullated” fibres of the sympathetic. A delicate membrane surrounds the white substance, called the “primitive sheath,” or the “sheath of Schwann” (S). Nuclei (n) lie at intervals within the sheath, between it and the myelin. The white substance is interrupted at regular distances by what are termed “nodes,” sometimes, from their discoverer, “nodes of Ranvier” (N). The end of each portion, or “internode,” is enclosed by the sheath, through which the axis-cylinder passes. Between the incurved extremities of the sheath is a little clear cementing substance, shown by the fine dotting in the figure. There is one nucleus (n) to each internode, about its middle. Around the nucleus is a little protoplasm, and it is probable that a very thin layer of protoplasm everywhere lies between the sheath and the myelin. Each internode may be conceived as a cell, a fat-cell, according to Ranvier, consisting of membrane, nucleus, protoplasm and fatty matter, the cells being arranged end to end, and the axis-cylinder passing through them, as a string passes through a series of tubular heads. The internodes are shorter towards the termination of a nerve. It is important not to confound the nodes with other, imperfect, divisions, sometimes termed the “incisions of Schmidt” (IS, Fig. 27). These are oblique, incomplete divisions of the white substance. Many exist in each node. They are believed by some histologists to be of artificial origin, but their uniform character seems conclusive evidence that they depend on structural conditions.

gists to be of artificial origin, but their uniform character seems conclusive evidence that they depend on structural conditions.

The myelin, Ranvier suggests, must protect the axis-cylinder, since its almost liquid consistence will diffuse pressure on the nerve. It may also have an insulating action, which, while not essential for conduction, may render conduction more perfect. The nodal segmentation keeps the almost liquid myelin uniformly distributed along the fibre, and permits nutrient material to reach the axis-cylinder.

The "grey fibres," "non-medullated fibres," consist of an axis-cylinder, sheath, and nuclei (Fig. 27 III), but contain no myelin. They constitute the sympathetic nerves, but some (probably sympathetic fibres) are found in all the spinal nerves. They are absent from the nerves of special sense, except the olfactory, which contains no other fibres.

The axis-cylinder of most nerve-fibres, probably of all, is continuous in one direction with the process of a nerve-cell, and may be regarded as the prolonged process of the cell, sharing all the changes of nutrition that the cell undergoes. It has been supposed that there are other centres for the nutrition of nerve-fibres than the cells from which they spring, but this theory, which rests on no direct evidence, is unneeded to explain the phenomena of disease.

The nerve-fibres are united into "fasciculi" by delicate nucleated connective tissue, and these fasciculi are similarly connected into larger bundles, while the whole nerve is surrounded by a dense, connective-tissue sheath. All these tracts of connective tissue are continuous. In them the blood-vessels run, and nerve-fibres are distributed. These *nervi nervorum* are derived from the nerve they supply.*

LESIONS OF NERVES.—*Secondary degeneration.*—As we have seen, a nerve-fibre undergoes the structural changes known as "degeneration" whenever it is separated from the cell with which it is connected. As a rule, the degeneration is in the direction of conduction, *i. e.* the cell from which the nerve-fibre conducts is that which governs its nutrition. An exception is presented by the sensory fibres in the peripheral nerves, which conduct upwards, but degenerate downwards when separated from the ganglia on their roots. The degeneration is commonly termed "secondary" because it is dependent on a primary lesion of another kind—as division of the nerve. Degeneration also follows many slighter lesions of a nerve, compression, over-extension, and the like, but it is not certain that this is always the same as that which follows a total lesion. The secondary degeneration is often called "Wallerian," from the name of the pathologist who first studied it. It is of great importance, practical and theoretical. The medullary sheath breaks up into segments, and these into smaller and smaller fragments, and the minute globules and granules are ultimately removed from the nerve-sheath. This is then empty, the axis-cylinder having also perished during the process. The nature of this process of destruction has been studied by Erb and others, but has been chiefly

* Horsley, 'Roy. Med. and Chir. Soc.,' January 22, 1885.

elucidated by the researches of Ranvier, and must be considered in some detail. Ranvier has shown that it is not a mere process of death or decay, but an active process, a destruction of the nerve as such by the protoplasm and nuclei of the internodal cells that constitute it.

The nature of the process has been chiefly studied in animals. The most important facts are illustrated in Fig. 28, in which the examples

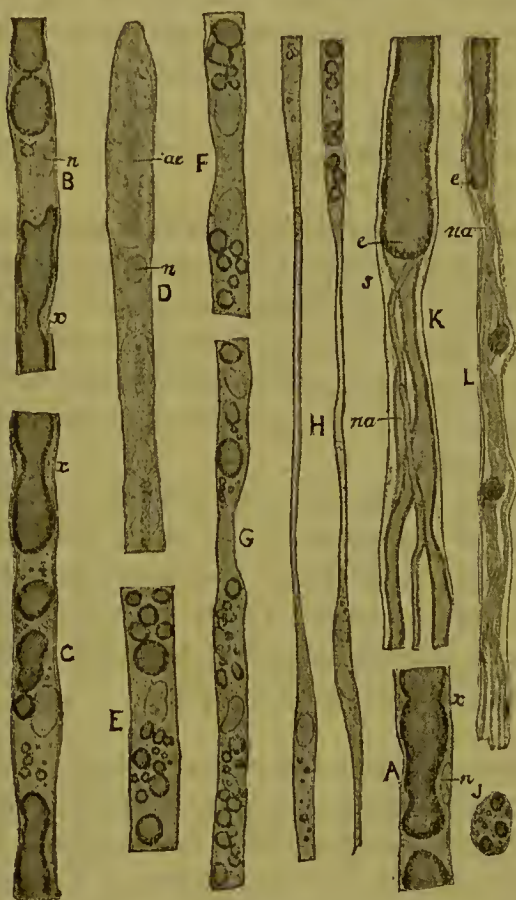


FIG. 28.—A–J. Degeneration of nerve-fibres (osmic acid and carmine staining). A, from sciatic of rabbit four days after section; B, C, same, fifty hours after section; D, a fibre stained with carmine only, to show the axis-cylinder; F, G, fibres from pigeon, three days after section; H, two fibres from pneumogastric of rabbit six days after section; J, a lymphatic cell from inter-fibrillar connective tissue, containing globules of myelin that it has taken up; *n, n*, nuclei; *x, x*, constrictions of the myelin produced by the growth of the protoplasm; *ac*, axis-cylinder. K, L, regeneration of nerve-fibres: K, from pneumogastric of rabbit, seventy-two days after section; L, from sciatic of rabbit ninety days after section; *e*, rounded end of white substance of central end of nerve; *s*, sheath; *na*, new axis-cylinder; in L are two globules of myelin remaining from the degeneration of the old fibre.

have been selected from Ranvier's figures and reduced to one tint. In the rabbit the first changes are to be perceived at the end of twenty-four hours. The nuclei are increased in size (Figs. A *n*, B *n*), the amount of protoplasm about them is greater than normal and it is granular, and there is in places a local increase in the amount of protoplasm within the sheath, compressing the myelin (A *x*, B *x*, C *x*). The nuclei then become detached from the sheath; the protoplasm everywhere increases, and encroaches on the myelin, until here and there it meets across the tube, completely separating the myelin (A, lower part; B, upper part) and with the myelin the axis-cylinder (D).

This process then goes on with increased rapidity; the myelin is broken up into smaller and smaller fragments (C, E, F) which are globular in the watery protoplasm, just as oil forms globules in water. The nuclei meanwhile continue to enlarge, and then divide, first the nucleolus and then the whole nucleus (F). The two nuclei may again divide, until (as in G) there are four or more nuclei in each internode, instead of one only as

in health. The small globules of fatty myelin seem to become changed in chemical composition, since they are stained less deeply by osmic

acid. Ranvier suggests that their fatty matter may undergo a process of saponification. Ultimately they seem to pass through the sheath, are taken up by connective-tissue cells and lymphatic cells in the vicinity (as in J), and are gradually, for the most part, removed. By the time the myelin is in small globules the nuclei cease to multiply. On the removal of the products of degeneration the sheath shrinks, and looks empty in places, but here and there it is enlarged by the nuclei, protoplasm, and a few remaining myelin globules (H). Hence in transverse section many small sheaths are seen with a few of larger size where they have been cut across at these swellings.

In the rabbit the first changes are visible at the end of twenty-four hours; the first complete interruption of the myelin and axis-cylinder occurs about the end of the second day; the process of destruction is considerably advanced at the end of the fourth day, and is finished, and the multiplication of the nuclei ceases, towards the end of the second week. The changes begin simultaneously in the whole extent of the separated portion of the nerve. They progress more rapidly at its peripheral extremity than elsewhere. At the end of the second day, in the rabbit, stimulation of the nerve by electricity no longer causes muscular contraction. Thus the disappearance of the electrical excitability coincides in time with the first complete segmentation of the myelin and axis-cylinder. This interruption explains what is termed the "loss of excitability;" it does not follow that the fragments of axis-cylinder cannot be stimulated; their stimulation can have no effect. When they cease to be *excitable* we cannot tell. But it is not certain that "loss of excitability" is only the result of loss of continuity. (See p. 42.)

Thus the process is the result of an active growth of the nuclei and protoplasm of the nerve, *i. e.* of the cellular elements of which the nerve is composed. Why should this occur? The determining cause is the interruption of the axis-cylinder—its separation from the cell of which it is a process. It follows equally destruction of the nerve-cell. Ranvier points out that the destructive exuberance of the protoplasm, which follows loss of function in the axis-cylinder, suggests that normally this function restrains the vital energy of the cell-elements. But it is possible that changes in the nutrition of the axis-cylinder precede the segmentation.

A process of destructive cell-activity suggests the idea of inflammation. The degeneration may be regarded as a process of parenchymatous inflammation. Several observers have described other indications of inflammation outside the fibres, especially increase of nuclei, and accumulation of leucocytes in the interstitial connective tissue, and even in the nerve-sheath, and also dilatation of the blood-vessels. Such changes are intense at the primary lesion. Their degree in the nerve below the lesion is very variable, and seems to be influenced, in some degree, by the irritative nature of the primary disease.

The process of secondary degeneration occurs more slowly in the

rabbit than in the bird, and seems to be still slower in man, in whom it is probable that complete segmentation does not occur until between the fourth and eighth days. It is certain that an identical process



FIG. 29.—Degenerating fibres from cutaneous nerves of man. (After Pitres and Vaillard.) A, from near a bed-sore in a case of fracture of the skull; B, C, D, from the fifth nerve in a case of neuralgia and ulceration of the lip; *n, n*, nuclei; in A the protoplasm and nuclei are increased, myelin is being broken up, the processes having proceeded furthest in the neighbourhood of the nucleus in the middle of the fibre; in B the segmentation has gone on to the formation of globules, which in C are, for the most part, small, and many have been removed, so that the fibre is narrow; while in D all the products of degeneration have been removed from considerable tracts of the sheath.

ings in the same degree. The evidence of this is that the faradaic irritability of the muscles (which depends on the nerves within them),

does occur in man. This is placed beyond doubt by the researches of Pitres and Vaillard on the changes in peripheral nerves in the neighbourhood of bedsores, &c. These are shown in Fig. 29, a comparison of which with Fig. 28 will show the identity of the process. It is highly probable that after complete division of a nerve in man the changes are the same as in animals. But the most common lesion in man is neuritis, and in this the process of degeneration is probably greatly modified by the severity of the primary lesion; in some cases it occurs rapidly, in others very slowly. In the latter cases, which are chiefly those of focal neuritis, the process cannot be the same as that which follows division of a nerve. The change in irritability, which will be more fully described in the account of the symptoms, is a slow depression, sometimes moderate in degree. The depression may be preceded by an increase in irritability. In such cases, there can be no complete segmentation of the nerve-fibres. There must be a gradual alteration in the molecular nutrition of the axis-cylinder, changing its excitability. Even in severe cases there is not usually a sudden loss of irritability; the current necessary for stimulation has to be made stronger and stronger, until at last the strongest current the patient can bear fails to cause muscular contraction. But we cannot infer from this that there is ever an actual interruption of the axis-cylinder. A stronger current might still excite the nerve. Moreover, as we shall presently see, when a nerve is being regenerated, an axis-cylinder may be continuous, and may even conduct, and still not be excitable.

All severe changes in the nutrition of the fibres involve the intra-muscular nerve-end-

presents changes quite similar to that of the nerve-trunk. But this is not always true in slight changes of nutrition of the nerve. We shall presently see that the slight alterations of irritability in the nerve and muscle do not always correspond. The nerves terminate in structures of special nature, and these may well have some slight degree of nutritional independence.

Regeneration may occur in the nerve after the degeneration is over. It is a slow process, occupying the second, third, and fourth month after division. According to Ranvier, it occurs always by the growth of new axis-cylinders from the central end of the nerve (see Fig. 28, K and L) which ultimately become covered with myelin. One or more new fibres may spring from each central fibre, and these may subdivide. All are enclosed in a sheath which is continuous with that of the central end (Fig. 28, K, s.). It is to be presumed that only some of these axis-cylinders persist and achieve functional permanence. Sometimes these fibres twist about, and even turn back and grow upwards, probably in the direction of least resistance. In animals, new fibres may grow through a considerable extent of cicatricial tissue between the divided ends of a nerve, but in man it is doubtful whether regeneration of a divided nerve occurs unless the extremities are brought in contact, or at least close proximity. Some investigators believe that there is a formation of fibres in the peripheral extremity, as well as a growth of new fibres from the central end. It is highly probable that the process, in the peripheral part of a nerve, is influenced by the connection with other nerves and the recurrent course of anastomosing fibres; because, in man, the suture of a nerve divided some time before, in the peripheral part of its course, has been followed by very rapid restoration of function. In cases of injury which does not involve an actual interruption of the nerve-fibres, regeneration occurs more readily, and very readily in slight lesions, in which it is probable, as we have just seen, that degeneration has been incomplete, and the continuity of the axis-cylinder has not been broken. The regenerated nerve-fibres regain some conducting power while they are still much narrower than normal, and before they can be excited by electricity.

Muscles.—The degeneration of the motor nerves is attended by changes in the nutrition of the muscles. These commence in or after the second week. The muscular fibres become narrower, and may be reduced, ultimately, to one-third of their former width. The transverse striation becomes less distinct, and the striæ seem to be nearer together than in health. The fibres may become slightly granular, but do not, as a rule, present actual fatty degeneration. If no regeneration of the nerve takes place, the transverse striation gradually disappears, and may be replaced by a longitudinal striation, or the fibres may undergo certain chemical changes, in consequence of which they present a peculiar glassy appearance, which has been called “vitreous degeneration.” During the progress of the changes in the fibres, the nuclei of the sarcolemma and of the interstitial tissue are increased in number,

and cellular elements, either newly formed, or migrated from the vessels, develop into fibres, so that ultimately the muscular fibres are separated by considerable tracts of connective tissue, and a state of cirrhosis results. If regeneration of the nerve occurs, the muscular changes are arrested, and the normal condition of the fibres is slowly restored. The amount of connective tissue in the muscle is often permanently increased, but the size of the fibres is reduced out of proportion to the increase in the connective tissue, so that the muscle is for a long time smaller than normal, and its natural bulk may never be regained. If no regeneration of the nerve occurs, the increased fibrous tissue takes the place of the lost muscular tissue, and slowly contracts, so that permanent shortening may result. Similar shortening sometimes occurs when there is partial recovery of the nerve and muscle. In most lesions of nerves, other than actual division, some fibres recover, even though others are permanently destroyed. The muscular degeneration is apparently the result of that of the motor nerves. All the phenomena can be thus accounted for. (See p. 21.)

SYMPTOMS OF NERVE-INJURY AND DEGENERATION.—The symptoms that attend the lesions of motor nerves and the consequent degeneration are of great importance. The lesion of the nerve causes paralysis of the muscles supplied by it, due to, and in proportion to, the interference with the conducting power of the nerve. The muscles at once become flabby from loss of tone, and to this atony actual wasting is added in the course of a few weeks. The wasting is due to the reduction in size of the muscular fibres. If the sensory nerve-fibres are not interrupted, the muscles become tender to the touch, and pain is caused by their strong contraction, due probably to the interstitial inflammation, and the morbid sensitiveness of the sensory nerves that end in the interstitial tissue.

The most important symptoms are those that are afforded by electrical examination of the muscles and nerves, since they enable the degenerative changes to be ascertained and followed during life. The alterations in the electrical reaction, consequent on this degeneration, have been already briefly mentioned (p. 19), but must now be described in greater detail. The rapid degeneration of a nerve, which follows a severe lesion, is attended by a loss of irritability on electrical stimulation, the loss being the same to faradaism and voltaism. After such lesions as are common in man, neuritis for instance, there is no sudden loss, such as occurs after injury of a nerve in an animal, when the nerve becomes segmented, but there is a more or less rapid diminution of excitability, and this goes on until no stimulation can be produced, even by a strong current. The progressive changes in irritability may be conveniently represented on a chart. Fig. 30 shows the typical course of the changes of irritability in a case of moderate severity. In the muscle (M) a fall of irritability (due to the degeneration in the

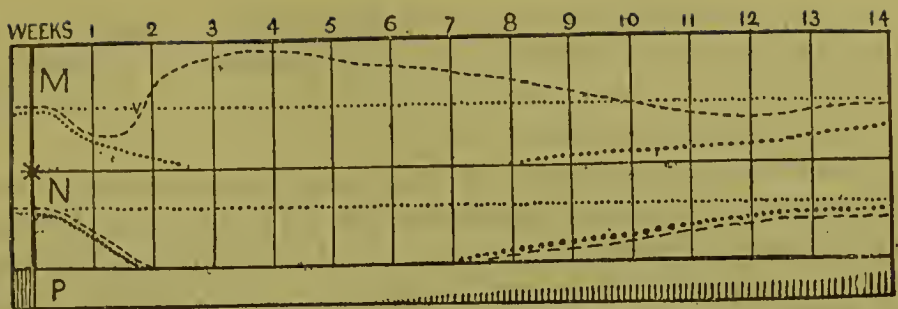


FIG. 30.—Type of degenerative reaction in a case of nerve-injury of moderate severity. (After Erb.) Muscle irritability lowered from middle of first week; faradaic, extinct in middle of third, reappearing in ninth; voltaic, increased from middle of second week until tenth, then depressed until fourteenth. Nerve irritability lowered from middle of first week, lost at end of second, reappearing at seventh. Power lost until end of fifth week.

In this and the following figures the normal degree of irritability (ascertained from the healthy side) is represented by the finely dotted horizontal line (*n.l.*); faradaic irritability, *F*, by a line of larger dots; voltaic irritability, *V*, by a broken line; *M*, muscle; *N*, nerve; *P*, power of voluntary contraction; the asterisk marks the occurrence of the lesion. The vertical lines represent time intervals.

nerve-endings) occurs simultaneously with that in the nerve-trunk (*N*) and the faradaic excitability becomes extinct at the same time in both nerve and muscle. The fall in voltaic irritability is quickly arrested by the change in the muscular fibres through which they soon become more excitable than normal to the voltaic current. This change usually occurs during the second week, and the irritability continues to increase during the third and fourth weeks. At its maximum it may amount to three, four, or five cells of the battery, *i. e.*, contraction can be obtained in the paralysed muscles with a current weaker, by so many cells, than is necessary to cause contraction in a corresponding unaffected part. The further course of the changes in irritability depends on the severity of the lesion and the intensity of the degeneration. In a case of moderate degree, such as is shown on the chart, nerve irritability reappears about the end of the second month, usually after some voluntary power is regained. It is at first low, so that a strong current is required. It gradually increases, but for a long time continues a little below the normal degree. This return of nerve irritability is accompanied by a corresponding return of faradaic irritability in the muscles (*i. e.* in the intra-muscular nerves). The increase of voltaic irritability often persists long after recovery of power, but it lessens as faradaic irritability returns, and, as shown in the chart, it may fall below the standard of health before it ultimately regains the normal degree.

Slight changes in irritability can be ascertained only by a comparison with the corresponding part on the other side in the same individual. Moreover, when we speak of excitability being "lost," we mean that we can obtain no stimulation by any strength of current that can be borne. This strength varies in different persons and in different parts. The more sensitive the part, the earlier irritability seems to be lost,

and the later it seems to return. In many cases, if we could use very strong currents, the irritability would be found to be merely much lowered, although it seems to be extinct when we can only test it with currents of moderate strength.

If the lesion is very severe, so that there is no recovery, and no regeneration of the nerve, the loss of nerve irritability, and of the faradaic muscular irritability, is permanent. The increase in voltaic irritability persists for months, and then gradually falls, as the muscular fibres waste, and becomes lower and lower (see Fig. 31)

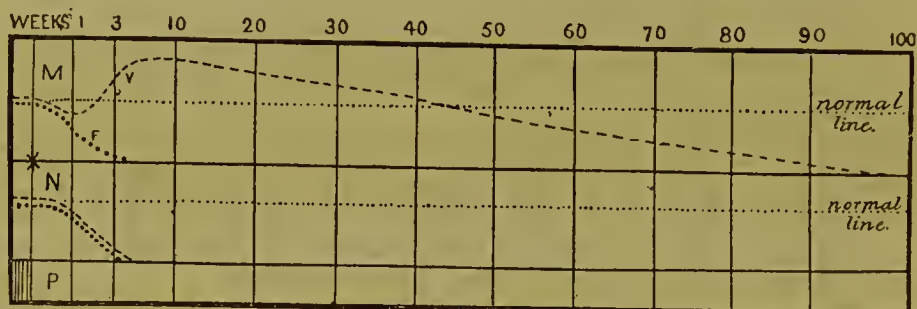


FIG. 31.—Type of reaction in a case of complete and permanent damage to a nerve. (After Erb.) Early course as in the last diagram; no return of power, nerve irritability, or faradaic irritability in the muscle. The early increase in voltaic irritability gradually lessens and at the end of ten months falls below the normal, but is not extinct until nearly two years.

until ultimately no reaction can be obtained, the fibres having perished. It does not become extinct until at least a year has elapsed, and sometimes (as in the chart) only towards the end of the second year. Often, when no contraction can be produced on the first attempt, after two or three separate applications of voltaism, distinct contractions are obtained.

The changes we have hitherto considered are in the *degree* of irritability, “quantitative” changes. But the quantitative increase in the muscular irritability is accompanied by a change in the order of response, according to the pole that is applied and the strength of current that is used—a “qualitative” change. We have already considered its general characters (p. 21), and have seen that it consists in an undue readiness of response at the positive pole (Anode) compared with that at the negative pole (Kathode) (Fig. 32, B and C), the muscle being normally the more sensitive to the latter (Fig. 32, A). Writing Cl. for the closure of the circuit, O. for its opening, and C. for contraction, the normal reaction is:

1. K.Cl.C; 2. $\begin{cases} \text{An.Cl.C.;} \\ \text{An.O.C.;} \end{cases}$; 3. K.O.C.; in disease we have
1. $\begin{cases} \text{K.Cl.C.;} \\ \text{An.Cl.C.;} \end{cases}$; 2. An.O.C.; 3. K.O.C.; or even
1. An.Cl.C.; 2. K.Cl.C.; 3. K.O.C.; 4. An.O.C.

Sometimes a slight abnormal tetanic contraction during the passage of the current also occurs.

This qualitative change is not always present, at any rate in recognisable degree. Even when there is a marked quantitative change, the kathodal closure contraction may still occur first. When the change is present, it is only in the muscles, and it must depend on the muscular fibres themselves. In the motor nerve the kathodal response is always the first, although a qualitative change has been detected in degenerated sensory nerves.

The muscular contractions which occur thus with undue readiness differ from normal contractions, excited through the nerves, in their distinctly deliberate character. Instead of the quick, lightning-like

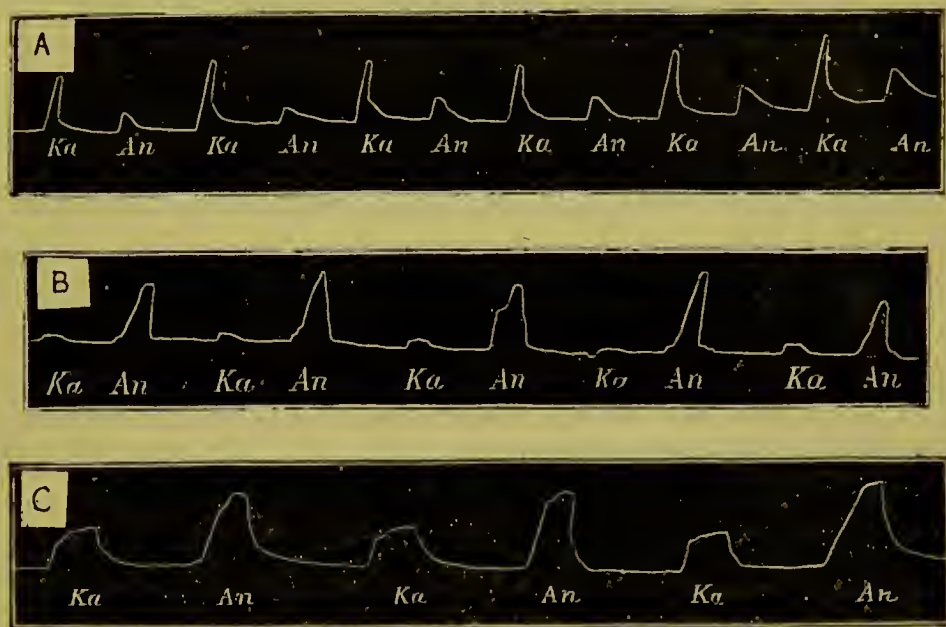


FIG. 32.—Tracings of the muscular contractions in nerve-degeneration. (After Erb.) *Ka*, Kathodal closure contraction; *An*, Anodal closure contraction. A, tracing in health; *Ka* much stronger than *An*; contractions sudden. B, tracing in nerve-degeneration with moderate current. *An* much greater than *Ka*, the latter scarcely visible. Contraction slower, shown by the more sloping upstroke. C, the same, with a stronger current. *Ka* greater, but still less than *An*; the slow character of the contraction and its long duration well marked.

contraction, the movement is distinctly longer in reaching its maximum and longer in its duration. This difference is very well shown in the accompanying tracings of contraction, after Erb (Fig. 32). During the period of increased voltaic irritability, the mechanical excitability of the fibres is often increased. If they are directly percussed, they respond with a distinct slow contraction (Erb).

Such are the changes in irritability which occur in cases of nerve-lesion and degeneration of moderate and considerable degrees of severity. Certain variations are occasionally met with and these are especially frequent cases of neuritis of slight degree. In severe cases the fall in nerve irritability, which usually commences in the middle of the first week, may not take place until the end of this week, although it may then progress so rapidly that no stimulation can be

obtained at the end of the second week. An example of this is shown in Fig. 33.* The same chart, and some of those that follow it, illustrate another very common variation from the type above described. There may be no fall in the voltaie excitability of the muscles before the commencement of the degenerative increase. Indeed this initial fall is as often absent as present.† The change in the nutrition of the muscle may then coincide with, instead of succeeding, the degene-

FIG. 33.



FIG. 34.

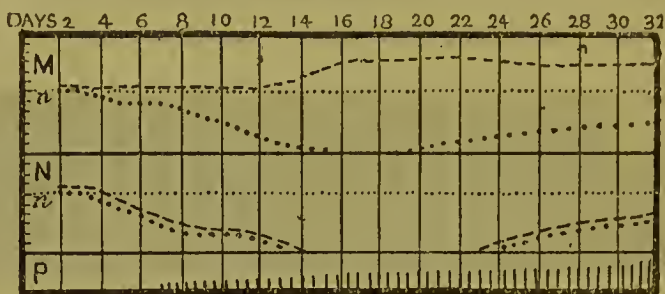


FIG. 33.—Severe neuritis, paralysis complete for months; recovery imperfect. Irritability of nerve, normal during first week; fell rapidly during the second; lost at its close. Simultaneous increase in voltaie irritability. The divisions on the left side represent cells of the voltaie battery, and half-centimetres of the secondary coil of Stöhrer's induction apparatus, in this and the following charts.

FIG. 34.—Slight neuritis; slight return of power on seventh day, slowly increasing; normal in fifth week. Muscle: faradaic irritability lessening from fourth day, lost on fifteenth, reappearing on twentieth. Voltaie irritability normal till twelfth day, then augmented. Nerve-trunk: lowered from fourth day without initial increase, reappearing on twenty-third day; changed alike to faradaism and voltaism.

ration of the nerve-endings (Figs. 33, 35). Sometimes, if the degeneration is rapid, there is an interval of a few days before the secondary changes in the muscle have reached the degree necessary to produce increased irritability. In a slight degree of degeneration, the increase in the voltaie irritability of the muscles may be postponed for a week or more after the nerve irritability begins to fall, and until the rise occurs, the voltaie irritability of the muscles may remain normal, even when their faradaic irritability falls with that of the nerve trunk. An instance of this is shown in Fig. 34.

When the nerve-lesion is very slight in degree the fall in nerve irritability may be preceded by a rise, which may be far greater in degree than the subsequent fall. The fall may indeed be not only slight, it may be altogether absent, so that the rise constitutes the only symptom. This initial increase in irritability is of considerable

* This and the following charts are from cases of neuritis of the facial nerve.

† The fall seems to suggest that the voltaie irritability of the muscular fibres is normally below that of the nerve-endings, but the fact that there is often no fall seems incompatible with this explanation. It may be that the effect of a very rapid degeneration of the nerve is first to lower the irritability of the muscular fibres, perhaps by an irritative inhibition, and it is only when this has passed away, by the greater degeneration of the nerve-endings, that the muscular excitability can be manifested. When the change in the nerve is slight such inhibitory influence may be absent.

interest as the manifestation of the slightest degree of alteration in the nutrition of nerve-fibres.* It may last for a few days or for two weeks (Fig. 35), and I have once known it to continue for five weeks (Fig. 36). Although the change in irritability of the nerve is usually the same to both currents, a partial exception to this rule is presented by the increase in irritability we are now considering. The change is not always quite equal to faradaism and to voltaism, and it is frequently much more marked to the isolated faradaic shocks, than it is to either the voltaic current or the rapid succession of shocks that

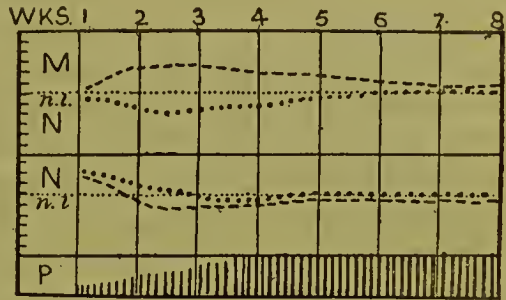


FIG. 35.—Slight neuritis; power not entirely lost, and became normal during the third week. Slight deg. react. in muscle developed during second week, and continuing till seventh. Nerve irritability increased during second week, passing during the third into transient depression, the voltaic irritability falling more than the faradaic.

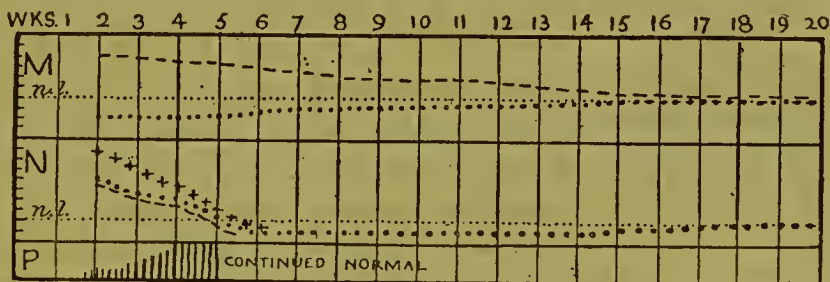


FIG. 36.—Slight neuritis; quick recovery of power during third and fourth weeks. Degen. react. in muscles present at the end of the second week, slowly lessening, but present in slight degree until the fourth month. Considerable increase in irritability in the nerve-trunk at the end of the second week, greatest to the faradaic shock (crosses) and lasting until the fifth week.

constitutes the faradaic current. This is shown very strikingly in Fig. 36, and in less degree in Fig. 38. In the former the irritability to the two currents was the same; in the latter that to voltaism was distinctly the greater. I have once met with a slight but distinct and prolonged diminution in faradaic irritability when no change could be found to voltaism (see Fig. 37). Bernhardt has observed lessened irritability to faradaism with distinct increase to voltaism in an ulnar nerve the seat of traumatic paralysis. In one singular case of double

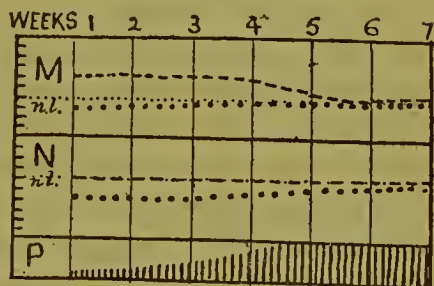


FIG. 37.—Slight neuritis; power regained during the second week. Deg. react. in muscle distinct on eighth day, lessening during second and third weeks. Nerve: lowered faradaic, normal voltaic irritability

* A similar change is met with in some central diseases, as chorea and paralysis agitans.

facial palsy, with weakness of the limbs, and changes in their muscular irritability, the voltaic excitability of the facial nerves was not lessened, although faradaic irritability was lost to such a current as could be borne. The case resembled in characters diphtheritic paralysis, although there was no history of diphtheria. It was probably either a form of multiple parenchymatous neuritis, or changed nutrition of the ganglion-cells. The symptoms gradually passed away.*

Moreover, the law that the faradaic irritability is changed in the same degree in the muscles (nerve-endings), and in the nerve-trunk, is also not always true of slight cases. The muscle-nerves may present no change when the excitability of the nerve-trunk is considerably increased (Fig. 38). The probable significance of this difference has been already mentioned (p. 43).

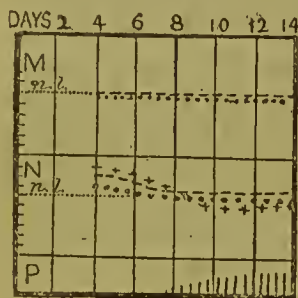


FIG. 38.—Very slight neuritis. No change in muscular irritability; irritability of nerve-trunk increased from fourth to eighth day, most to faradaic shock (crosses), least to faradaic current.

The electrical excitability of a nerve and its power of conduction are usually lost together. But when a nerve is recovering from a local lesion, and regeneration is in progress below the lesion, some power of conducting the voluntary stimulus may return in the peripheral portion before it becomes excitable by any form of electricity. Another rare anomalous condition has been noted by Bernhardt and Grünberg, in recent cases of nerve injury. When the lesion arrests all conduction of the voluntary stimulus a strong faradaic stimulation of the nerve above the lesion may still cause slight contraction in the paralysed muscles. It is probable that this difference is simply one of strength of stimulus, and that the nerve impulse generated by the electricity is stronger than that descending from the brain.

Recovery of the nerve is attended by gradual restoration of power over the muscles, the nutrition of which slowly improves; but if the wasting has been considerable, a long time elapses before they regain their normal size, and, as already stated, they may be always somewhat smaller than the corresponding muscles on the unaffected side. Curious secondary spasmodic symptoms are common after palsy of the facial nerve (q. v.)—spontaneous muscular contractions, and associated over-action of the affected muscles. The associated over-action is very rarely met with after palsy of the nerves of the limbs, but coarse, fibrillary, muscular contractions are not uncommon, and may persist for years after recovery. Their probable nature is discussed in the account of the pathology of facial paralysis.

Sensory symptoms also result from lesions of the nerves that contain

* The observations were made with great care and with full knowledge of a frequent source of fallacy—the diffusion of the voltaic current to the muscles themselves. Cyon has observed a similar change after injury to a nerve in an animal.

sensory fibres. The interruption of the fibres arrests conduction, and causes loss of sensation in the part supplied by the nerve, just as it causes motor palsy. But a slight lesion of the nerve may cause persistent muscular paralysis, and sensation may be unaffected, or impaired only in slight degree, and for a short time. This result is so frequent that we cannot ascribe it to a difference in the relative damage to the motor and sensory fibres. The probable explanation is that a slight degree of conduction may suffice for the stimulation of the sensory centres in the brain, and not for that of the muscular fibres. In another class of cases, however, we must seek a different explanation of the persistence of sensibility in the area supplied by the nerve. There may be no loss even when a nerve is completely divided. This is observed chiefly near the extremity of a limb. It can only be explained by what is termed "recurrent sensibility." Anastomoses exist between the terminal fibres of different nerve-trunks, and it would seem that, in some individuals, the connection is so abundant as to suffice for conduction. Thus, of two persons in whom the ulnar nerve at the wrist has been divided, one may have total anæsthesia in the fingers supplied by the nerve, and in the other there may be little or no loss.

The peripheral segments of the sensory fibres degenerate, like the motor nerves. We cannot test them, and ascertain the degeneration in the same way, in severe lesions, because their stimulation cannot be perceived. Nevertheless, an altered polar reaction has been observed in slight cases, analogous to that which occurs in the muscles. A sensation is produced with the positive pole by a weaker current than is required with the negative pole. In very rare cases of nerve injury a delay in conduction of pain has been observed.

Increased sensitiveness in the area of distribution of the nerve, with or without spontaneous pain, is very common in cases of partial lesions of nerves. It must be referred to the morbid changes in their fibres, alterations in the nutrition of the axis-cylinder, probably analogous to the slight changes in the motor nerves. Hyperæsthesia, increased sensory excitability, is analogous to the increased motor excitability that we have seen is sometimes present. Pain and tenderness of the nerve-trunk are also frequent in the same cases, due no doubt to the increased sensitiveness of the "nervi nervorum" distributed in the sheath. The pains of both kinds are sometimes very severe and persistent.

Reflex Action.—All lesions of nerves abolish reflex action in the area of the distribution of the nerve affected. In the muscles supplied, myotatic irritability is also lost, and this loss may persist long after the other symptoms have passed away.

Vaso-motor and trophic disturbance occurs frequently in consequence of lesions of nerves. The vaso-motor nerves run in the mixed nerve-trunks, and suffer with the other fibres. The general character of the disturbance has been already described. When the lesion is acute and

irritative, vascular dilatation and an increase of temperature occur at first, and are followed by passive hyperæmia and lowered temperature. There may be œdema, and an increased secretion of sweat. The changes in the skin, acute and chronic, are those that have been already described (p. 18). They may occur when there is no conspicuous evidence of vaso-motor paralysis. The atrophy and smoothness of the skin is especially common. Occasionally, instead of becoming thin, it increases in thickness, the nails often become thick and rough and present transverse furrows. The growth of the hair may be lessened or increased. Sloughing of the skin is far less common than in central diseases, but vesication occurs with extreme readiness. Mustard plasters also blister the skin more readily than in health. It is important to remember this, because the affection is one in which hot applications are often recommended. A gentleman dislocated his shoulder, and either by the displaced bone, or in the reduction, the brachial plexus was seriously injured. The muscles of the hand wasted, and the skin became glossy. He was advised to bathe the hand daily in hot water. One day his wife bathed it in water which to her was pleasantly warm, but the result was that the hand was covered with blisters, and ulcers were left which did not heal for months. The joints often suffer in their nutrition. Acute inflammation, such as occurs sometimes in spinal affections, is rare, but chronic changes in the joints are frequent. Adhesions and alterations in the articular surfaces often limit the movement of the joints. When the muscular wasting is slight, the condition may resemble a primary joint affection, and thus a mistake in diagnosis is sometimes made.

INFLAMMATION OF NERVES: NEURITIS.

Neuritis, or inflammation of nerves, presents various characters, which have led to the distinction of numerous forms. The inflammation usually affects chiefly the outer sheath of the nerve, and this has been rather needlessly distinguished as "perineuritis." It may involve chiefly the connective tissue between the bundles of nerve-fibres ("interstitial neuritis"); or the nerve-fibres themselves ("parenchymatous or degenerative neuritis"). Our knowledge of the latter is recent, but as an acute and chronic process it has been found to be much more frequent than was suspected a few years ago. Some forms of neuritis have a tendency to ascend the nerve, and this variety has been termed "ascending neuritis" or "neuritis migrans." Usually only one nerve-trunk is primarily affected, but sometimes many nerves suffer at the same time. This condition, "primary multiple neuritis," is of great importance, and will be described in a separate section.

Most forms of neuritis may be either acute or chronic. Acute neuritis usually subsides into a chronic stage, and its symptoms may thus persist for a long time. Other varieties that have been distinguished depend upon the cause to which the inflammation is secondary. Thus we may have cancerous, syphilitic, and other forms, and these distinctions have some justification in the fact that there are some pathological differences in these forms of secondary neuritis.

CAUSES.—Any injury to a nerve causes more or less traumatic inflammation at the seat of injury. Such inflammation occurs readily, and follows damage that is slight as well as that which is severe. Neuritis may thus be set up by all sorts of incised and punctured wounds, by contusions, and by over-extension of the nerves. Those that pass by joints are liable to injury from dislocations, either by the displacement of the bones, or from pressure during reduction. The brachial plexus often suffers from this cause. In fractures the nerves may be injured directly, or may be compressed by the callus that is formed. External pressure on the nerve may also produce inflammation; the musculo-spiral and the sciatic often suffer from this cause. The musculo-spiral and the nerve to the serratus are sometimes damaged by a violent contraction of the muscles through which they pass.

Neuritis may be set up by extension from adjacent inflammation. The nerves near suppurating joints may be involved, and even those that pass by a joint that is the seat of rheumatic inflammation. The intercostal nerves have been affected by extension from an inflamed pleura. The cranial nerves and the spinal nerve-roots are involved by extension with especial frequency. Some of the most important symptoms of meningitis are due to the invasion of the nerves.

Exposure to cold is another cause of neuritis. It is then often called "rheumatic." The nerve, or rather its sheath, may be primarily affected, or the first effect may be inflammation of the fasciæ, from which the inflammation may spread to the nerve-sheath. Persons who are liable to muscular rheumatism, and those who are gouty, suffer thus more than those who are prone to acute articular rheumatism.

Neuritis occurs also from certain general diseases. The chief are gout—from which it may develop without exposure to cold—syphilis, and cancer. In the latter it occurs only in the neighbourhood of a cancerous growth, but the inflammation may be apparently simple. It occurs rarely in leucocythæmia, from an infiltration of the nerve with leucocytes. Acute diseases may also lead to neuritis. Small-pox, typhoid fever, and others, may cause simple neuritis. Diphtheria causes a special "parenchymatous" form. Acute degeneration of the nerves has been found in the neighbourhood of bedsores in hemiplegia (Pitres). Such pressure-neuritis may be the mechanism by which bedsores are produced. Neuritis may result from lead-poisoning, although it is not certain that the characteristic paralysis which lead produces is due to neuritis. Lastly, multiple neuritis has been lately proved to

occur from many causes, of which the chief is alcoholism, and to form part of many diseases in which it was previously unsuspected.

Pathological Anatomy.—In acute inflammation the affected part of the nerve is red and swollen. The redness depends on distended vessels, which may be visible on the surface. The swelling is due to oedema, or to a sero-fibrinous exudation, sometimes jelly-like in aspect. The microscope shows

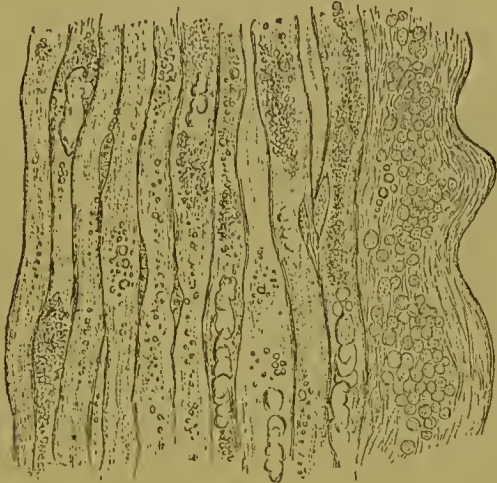


FIG. 39.—Neuritis: degeneration of nerve-fibres, the myelin broken up into masses, globules, and granules. Accumulation of leucocytes in nerve-sheath. From a case of multiple neuritis. (After Leyden.)

leucocyte-like corpuscles, surrounding the vessels, infiltrating the sheath (Fig. 39) and accumulating between it and the nerve. There may be even small extravasations of blood. These changes may be limited to the sheath, in what is called "perineuritis," or may extend into the substance of the nerve in "interstitial neuritis." In the latter case the lymphoid corpuscles infiltrate the septa, and may even be seen in the substance of the fasciculi between the nerve-fibres. These changes may be continuous along a con-

siderable tract of the nerve (diffuse neuritis), but more frequently they are chiefly marked at certain places, which are separated by portions of the nerve which are so little affected as to appear to the naked eye to be normal (focal or disseminated neuritis). The foci of inflammation are chiefly situated at places where the nerve turns round a bone, or emerges from canals or fasciæ, or divides.

The extent to which the nerve-fibres suffer varies much. They usually present little change, when the inflammation is limited to the sheath, unless the nerve lies in a bony canal, or in rigid fibrous tissue, within which the sheath cannot expand; its swelling then exerts pressure on the fibres. When the inflammation is interstitial the fibres suffer more readily, although not invariably. On the other hand, they are sometimes found much altered, when the connective-tissue elements are but little affected. In this case the inflammation is probably "parenchymatous" and begins in the nerve-fibres, the interstitial tissue being secondarily involved. The changes in the fibres are almost the same as those that occur in degeneration (Figs. 28 and 29). It was pointed out how closely the two processes of inflammation and degeneration are connected, and how difficult it is to separate, or even to distinguish them, in many cases. The myelin of the white substance first breaks up into segments, more or less elongated, often with smaller globules of myelin between or beside them. The masses

are cloudy or finely granular in aspect. The axis-cylinders are interrupted where the myelin is divided. The nuclei of the sheath are increased in number, and the protoplasm around them is increased in quantity. Next the myelin is divided into smaller globules and granules, and the axis-cylinder is no longer recognisable. The myelin then disappears in parts of the tubes, while it remains at other parts, but is still more finely divided. Lastly, the sheaths become empty, and very narrow, containing only nuclei at intervals, with here and there a little finely granular material, or sometimes some brownish pigment granules. The process may thus go on to complete destruction of the fibres. These commonly suffer unequally; fibres that have a normal appearance are scattered among those that are much altered.

In interstitial neuritis the axis-cylinders often suffer less than the white substance. The latter becomes atrophied, so that the fibres are smaller than normal. If the interstitial inflammation is very intense the fibres may break up as above described. As the inflammation subsides, the new cellular elements that have made their appearance assume the aspect of fusiform cells, and fibrous tissue is developed, either from these cells, which become less numerous, or from the intercellular exudation. This fibrous tissue surrounds and encloses the fasciculi, a condition that has been termed "sclerosis of the nerve." A firm fusiform swelling may remain at the affected spot, and this may be adherent to adjacent structures. Sometimes fat is ultimately formed in the new connective tissue, a condition that has been rather unnecessarily termed "lipomatous neuritis" (Leyden); the fatty deposit does not seem to be related to any special form of neuritis.

In syphilitic neuritis, which affects chiefly the cranial nerves, there is a cellular growth in the sheath and interstitial tissue similar to that which constitutes other syphilitic new formations, with a variable amount of leucocytal infiltration of simpler inflammatory character. The growth in the sheath may amount to a distinct syphilitic tumour, with or without interstitial changes. In cancer, nerves adjacent to the new growth may present simple interstitial neuritis or may be infiltrated by a growth of cancer-elements spreading to the nerve by direct extension.

The destructive changes in the nerve-fibres extend down the nerve to the periphery in a process of secondary degeneration, more or less inflammatory in nature, and accompanied by more or less interstitial inflammation. The changes that result are those that have been already described as occurring in secondary degeneration. Usually the central portion of the nerve remains free, the alterations ceasing a short distance above the seat of the primary inflammation. In rare cases an ascending neuritis (*n. migrans*) passes up the nerve, and may spread, at a plexus, to other nerve-bundles, and other nerve-trunks. The formation of fibrous tissue in the inflamed part, and the cicatricial contraction of this tissue, often prolongs the irritation

of the nerve-fibres, and the symptoms due to this, and a state of "chronic neuritis" is said to be left.

SYMPTOMS.—The symptoms of neuritis vary extremely according to its intensity, its extent, and the nerve that is affected. The onset of the acute form is sometimes attended by some constitutional disturbance, especially when many nerves are affected (see "Multiple Neuritis"). The chief symptoms are local. The most prominent is pain, felt in the inflamed part of the nerve, and also often in the part to which it is distributed. The local pain is due to the irritation of the "nervi nervorum," the distant pain is produced by irritation of the proper fibres of the nerve. Sometimes the pain involves the whole limb, and in severe cases it may be most intense, burning, boring, rarely darting, in character; usually worse at night; increased by movement, by postures that involve tension or pressure on the nerve, and by whatever causes passive congestion of the limb, such as the act of coughing. Sometimes it seems to radiate into distant parts, and not rarely pain is also felt in the corresponding region of the opposite limb. The sensitiveness of the whole of the affected region is increased, and even the bone may be tender, so that at first attention may not be directed to the nerve, but when this is pressed great pain is always produced. In slighter cases the pain is usually limited to the nerve and its distribution. If the nerve is accessible to direct examination it may be felt to be distinctly swollen at the affected part. Occasionally the skin over it has been observed to be red, and rarely there has been slight œdema. Spontaneous sensations may be felt in the region supplied by the nerve, tingling, &c., and the skin may be hyperæsthetic. After a time, as the nerve-fibres suffer, sensation may be perverted, or even lessened; complete anæsthesia is usually confined to small areas. The muscles supplied by the nerve become weak, tender, and present fibrillar twitchings, while they may contract occasionally in very painful cramp. Increased perspiration has been observed in the part of the skin supplied by the inflamed nerve, and sometimes, although rarely, eruptions occur. Herpes is not common from ordinary acute neuritis; a curious fact, considering that one form of neuritis seems to be its constant cause. Effusion into joints has been observed in very rare cases. The constitutional disturbance, which may attend the onset, subsides in the course of a few days, but the pain and other symptoms usually persist in undiminished severity for several weeks. They ultimately slowly subside into a chronic stage.

When the affection is chronic from the commencement, initial constitutional disturbance is absent. Pain is the prominent symptom from the first. It may preponderate either in the nerve at the seat of the inflammation, or in the region of its distribution. The affection of sensibility, and the trophic changes, are similar to those met with in the acute form. In both forms the muscles may waste, and present the reaction of degeneration when they are tested with electricity (p. 45). In slight cases the initial increase of excitability in the nerve is often

well marked. Trophic changes in the skin are very common, especially the chronic impairment of nutrition that results in the glossy condition. Slight alteration in the nutrition of the joints is also common, and adhesions form, limiting the movement, and fixing the parts in the position corresponding to the muscular paralysis.

When neuritis ascends a nerve, the symptoms gradually extend in area, and if it reaches the plexus from which the nerve proceeds, they may extend to all the nerves of the limb. This "migrating neuritis" is not rare in man, and is readily produced in animals. It may reach the spinal cord, and there produce various disturbances. The inflammation may spread in the tissue outside the dura mater, or may extend to the cord, and cause subacute or chronic myelitis with or without meningitis. The paralysis that occurs secondarily to some visceral diseases, as those of the bladder, and commonly regarded as reflex paralysis, are probably produced through the agency of an ascending neuritis. Lastly, the inflammation may extend to the nerves of the other side, usually to those that correspond to the primary seat of the disease. Such extension may be through the spinal cord or the membranes, but in some cases symptoms occur in the nerve of the opposite side, without any indication of an affection of the centres. Experiments on animals have demonstrated that such implication of the opposite nerve may occur when the centres are unaffected. It has been called "sympathetic neuritis." We have seen that reflected pains are occasionally felt in the corresponding nerve on the opposite side, and it is probable that, as in neuralgia, vaso-motor disturbance in the nerve-sheath may accompany such pains, and, in a predisposed person, may cause actual neuritis.

The *duration and course* of neuritis vary much. A slight acute neuritis may run its course in a few weeks and then subside. Much more commonly the affection persists in a chronic stage for many weeks, or even months, and slowly passes off. The "rheumatic" forms are as a rule much more tedious than those that result from injury, although traumatic neuritis is sometimes very insidious and may last a long time; it may ascend the nerve from its starting-place, and become localised in certain situations, where it may develop afresh, and give rise to symptoms that may seem to be independent of their cause.

DIAGNOSIS.—The diagnosis of neuritis depends, first, on the localisation of the symptoms to the distribution of a certain nerve-trunk, and secondly on the pain and tenderness in the nerve. The diffuse pains that attend the onset may be readily mistaken for the pains of acute rheumatism, or for those due to an inflammation of the bone, but in the course of a day or two the localisation of the symptoms declares their nature. The chronic form is easily mistaken for neuralgia, and the diagnosis is often difficult, the more so in that many so-called neuralgias are really due to neuritis. The distinction can only be fully discussed when we have considered the symptoms of neuralgia, but it

rests chiefly on the fact that in neuralgia the pain intermits more completely than in neuritis, there is not the same initial tenderness in the nerve-trunks, and the tender spots have a more uniform localisation. Lessened sensibility, showing organic damage to the nerve-fibres, is conclusive evidence of neuritis. The pains in some central diseases, chiefly those of the spinal cord, may be thought to be due to neuritis, but there is not the local tenderness of the nerve-trunk, and the pain is not limited to the distribution of a single nerve. As we shall see presently, the diagnosis of multiple neuritis from affections of the cord is far more difficult.

PROGNOSIS.—The gravest form of simple neuritis is that in which the nerve is affected secondarily to a local suppurative inflammation. The prognosis is best, as a rule, in traumatic neuritis, but the rule is one to which the exceptions are occasionally very considerable in degree. The rheumatic form is usually less grave than the traumatic. In all cases the intensity of the symptoms, and the evidence of descending degeneration, furnish a more trustworthy guide to prognosis than does the mere form of the affection. The effects of complete degeneration always endure for some months. Regeneration does not occur until the original cause has ceased to act, and then occupies many weeks in its progress to the restoration of functional competence. In all forms pain is apt to linger on, and in the last third of life it may continue for years.

TREATMENT.—The first consideration in the treatment of neuritis is the removal of its cause, if this can be discovered. A wound or injury or local inflammation adjacent to the nerve must be dealt with by appropriate measures. Any constitutional cause, such as gout, must be treated. For the inflammation of the nerve, it is of the first importance to secure to the part as perfect rest as possible. Movement causes mechanical irritation of the nerve, and involves functional stimulation of its fibres, both of which are injurious. The nerves of a limb run between muscles, and the contraction of these muscles causes pressure on the inflamed nerve, and irritates it, as the pain thus produced sufficiently shows. Pain is indeed a useful indication of the harmful influence of movement, and all movement that causes pain should be avoided. The posture of the limb should be such as to involve no pressure on or tension of the nerve. The general treatment of an acute neuritis must be that suitable for any acute local inflammation, whatever its seat,—an unstimulating diet, an aperient, and diuretics. In gouty cases a brisk purgative may, with advantage, be given. General diaphoresis is useful in cases that are due to cold, and in others local sweating is often of distinct service. The limb may be steamed, or exposed to hot air. This should be followed by hot fomentations applied along the course of the affected nerve, and these by linseed-meal poultices. Leeches may also be employed at the onset of severe cases. When the inflammation results from injury, cold may be applied along the course of the nerve instead of heat; both probably modify the vascular dis-

turbance of inflammation in a similar manner. Counter-irritation may be used at the onset of slight cases, but when the inflammation is severe, this agent is more effective during the subsidence of the inflammation than during its active stage. Blisters, repeated mustard plasters, or stimulating liniments may be used, but care must be taken not to blister skin that is anæsthetic, or troublesome ulceration may be caused. The same caution is necessary in regard to hot applications, as is shown by the case mentioned on p. 52.

Spontaneous pain requires sedatives, of which the hypodermic injection of morphia is by far the most effective. It should be used only for spontaneous pain, and not to enable the patient to use the limb in a way that would produce pain if morphia were not given. Mechanical irritation may be equally injurious, although the pain which it would cause is obviated by the sedative. An injection of cocain, $\frac{1}{8}$ — $\frac{2}{3}$ gr., also often relieves the pain. In the rheumatic form salicylate of soda has been given, but is of doubtful value. Iodide of potassium sometimes seems to be useful, but no agent has so distinct an influence on the process of inflammation in the nerve as small doses of mercury. A grain of blue pill may be given once or twice a day, and if morphia has to be injected at the same time, the mercury is useful also in correcting the constipating influence of the morphia.

In the chronic stage or form, counter-irritation, by blisters or cauterly, is of great value. So also is electricity, which has little influence during the acute stage. The voltaic current should alone be used. The positive electrode may be placed over the inflamed part of the nerve, or over the seat of pain, and kept there for ten minutes at a time, the strength of current being slight, such as the patient can just perceive. In very chronic cases a stronger current, sufficient to cause actual pain, is of service, applied in a similar manner, but for a shorter time. It has probably chiefly a counter-irritant influence. Faradism may be used in the same way. All painful impressions on the skin lessen, for a time, the nerve pain.

The muscles supplied by the inflamed nerve are best left alone, unless their wasting is marked, or the degenerative reaction shows serious damage to the motor fibres. In cases of moderate severity they will recover when the nerve recovers. All that is desirable is that they should be gently rubbed once or twice a day. If the wasting is considerable, however, or there is degenerative reaction, they should be stimulated to gentle contraction by a weak interrupted voltaic current. They should on no account be faradised during the active stage of the affection, even if they act to faradism. The acute pain that the faradaic stimulus causes, and the increased tenderness that lasts for hours afterwards, are sufficient evidence of its injurious effect.

In all cases attention to the general health is of great importance. Tonics are needed during the chronic stage. Change of air will sometimes remove, in a few weeks, symptoms that have previously been stationary for months.

The modifications of treatment that are rendered necessary by the position of the neuritis will be considered when we come to speak of the affections of the several nerves. The pathological varieties of neuritis only need special treatment in so far as they depend upon special causes, and this point, as already mentioned, must always be one of the first considerations.

A description of "multiple neuritis," which should properly be considered next, will be better understood after the symptoms of the disease of special nerves have been described.

MORBID GROWTHS IN NERVES: NEUROMA.

The term "neuroma" has been applied indiscriminately to all morbid growths situated on the peripheral nerves. The discovery, by Virchow, that many of these consist of a growth of nerve-fibres, while others consist of heterologous tissue such as constitutes morbid growths elsewhere, has led to the distinction of the former as "true neuromata" and the latter as "false neuromata," or "pseudo-neuromata," and even to the limitation of the term "neuroma" to a growth of nerve-tissue, whether in the central or peripheral nervous system, the heterologous growths being called by the names they bear in other situations, as "fibroma," "sarcoma," &c. The latter system of nomenclature, although certainly more consistent, has not become current in this country.

The true neuromata may consist of medullated or of non-medullated nerve-fibres, termed "myelinic" and "amyelinic" forms by Virchow. The latter were for a long time regarded as fibrous. The occurrence of ganglion-cells has been proved in only one or two instances. There is connective tissue between the nerve-fibres, which varies in amount and in character, and hence the firmness of these tumours also varies. This interstitial tissue may be so abundant as to constitute an intermediate form between the true and false varieties. This is probably the condition in most cases of multiple neuroma. The "false neuromata" may be of various nature, but fibrous growths, fibromata, are far more common than any other kind. Myxoma occasionally occurs, the new mucoid tissue growing from the nerve-sheath. Glioma is very rare on the peripheral nerves, although occasionally found on the auditory nerve. Various forms of sarcoma have been met with. Carcinoma also occurs, very rarely as an isolated growth, but not uncommonly as a more or less diffused or nodular infiltration of the nerve, arising by extension from a contiguous growth. Syphilitic

growths are common on the cranial nerves within the skull, but are rare elsewhere. In *lepra anæsthetica* the nerves are infiltrated with fibrous tissue, enlarging them to many times their normal size. The enlargement is rarely nodular, and is perhaps rather a chronic cirrhotic inflammation than a growth. (See Multiple neuritis.)

A curious variety of neuroma, consisting of interlacing cords, more or less nodular and tortuous, is termed *plexiform neuroma*. About twenty cases have been recorded. The disease commonly begins in foetal life, and is most common on some branch of the fifth nerve in the orbit or the upper eyelid, but has been met with over the temporal bone, in the lumbar, cervical, brachial, and solar plexuses, on the penis and the mamma. It may be quite superficial or deeply seated. The cords, from 1 mm.

to several centimetres in diameter, consist of a clear outer zone of concentric fibrillar connective tissue, of inner looser nucleated tissue, and, in the middle, a bundle of nerve-fibres, some normal, others degenerating. The cords are connected together by



FIG. 40.—Plexiform neuroma from the orbit (after Marchand). The connective tissue surrounding the cords has been removed except at *a*, and irregular, cylindrical, nodular cords are seen anastomosing. A nerve passes into the tumour and suddenly becomes enlarged.

loose tissue, sometimes myxomatous, separated from the proper tissue of the cords by an epitheliated space. The growth of this form is extremely slow, but it may exert compression on adjacent structures.* A remarkable case of extensive myxomatous disease of the nerves of the forearm, congenital in origin, presenting analogies to plexiform neuroma, is recorded by Mr. De Morgan.†

The subcutaneous extremities of sensory nerves are sometimes enlarged into minute tumours which, when painful, have been termed “*tubercula dolorosa*.” Neuromata in the skin may co-exist with similar tumours on the nerve-trunks. Those shown in Fig. 41 are from the same case as the tumours represented in Figs. 42—44.

The size attained by growths on nerves varies from that of a child's head to a nodule only just visible. They rarely exceed the dimensions of the closed fist. The variation



FIG. 41.—Cutaneous neuromata from skin of abdomen. (After Smith.)

* Marchand, ‘Virchow's Archiv,’ Bd. 76, p. 36.

† ‘Path. Trans.,’ vol. xxvi, p. 2.

FIG. 42.

FIG. 43.

FIG. 44.

FIG. 45.

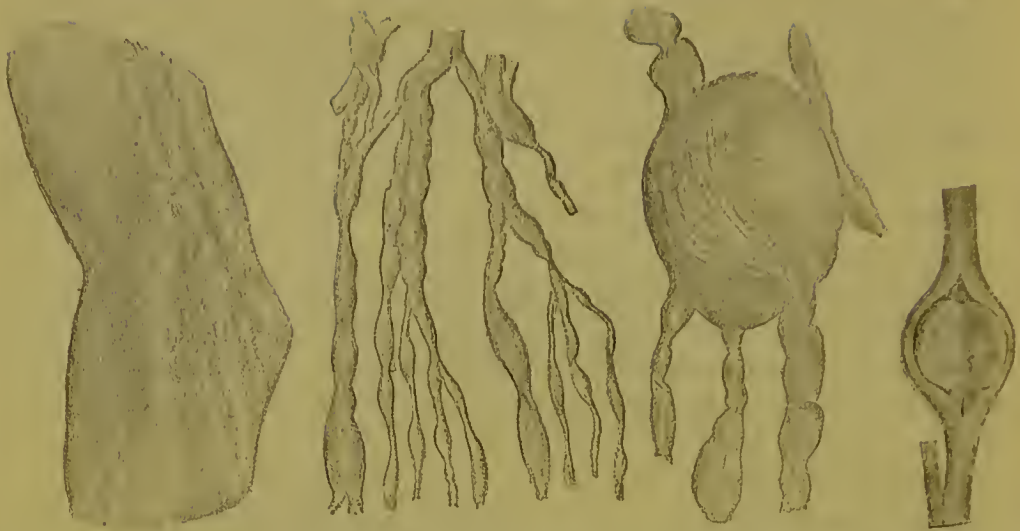


FIG. 42.—Right elbow of a man, æt. 30, with multiple neuromata; those on the nerves of the arm are visible beneath the skin as moniliform series of swellings along the course of the nerves. (After Smith.)

FIG. 43.—Part of the brachial plexus and nerves of the arm.

FIG. 44.—Neuroma of the posterior tibial nerve, same case. The tumour was the size of a lemon, and could be felt in the popliteal space. The flattened bundles of fibres of the nerve were separated and passed over the surface of the tumour. The portion of nerve to the right is a piece of the external popliteal. The tumours caused no interference with function. The patient died of enteric fever. One large tumour filled the pelvis.

FIG. 45.—Small neuroma of popliteal nerve laid open. It is seen to lie within the sheath. It caused no symptoms during life. (After Smith.)

in size of multiple neuromata is illustrated in Figs. 42—44, after Smith.* The large tumour (Fig. 42) was the size of a lemon, but in the same case a growth from the sciatic plexus almost filled the pelvis. They may occur on any nerve in the body, whether cerebro-spinal or sympathetic. False neuromata are generally single. When true neuromata are “multiple” they are often extremely numerous, and almost every nerve in the body may be transformed into a chain of growths. In the case figured more than 200 were counted in the right arm alone, and the total number of growths in the body cannot have been less than 1000.

The tumours are almost always within the sheath of the nerve (Fig. 45). Sometimes they are on one side, and the nerve may pass by unchanged. More often the substance of the nerve is involved, and the fibres may be separated and spread out on the surface (Fig. 44). Even then they may not be damaged. They suffer far more in heterologous growths than in true neuromata.

CAUSES.—The causes of neuroma are generally obscure. Multiple neuromata are sometimes hereditary and are probably due, in most cases, to a congenital tendency of tissue growth. They are said to be sometimes the result of general neurotic predisposition, and evidence of this is found in the occasional occurrence of neuromata in

* R. W. Smith, ‘Treatise on Neuroma,’ 1849.

the subjects of cretinism or iodicy, of which some remarkable examples have been recorded. Multiple neuromata are said to be almost confined to men. The isolated form is not uncommon in women. They may occur at any age. Virchow believes that they are unusually common in the phthisical and scrofulous.

Of traceable immediate causes the most frequent is traumatic injury. Pressure, punctured wounds, or division of the nerves may give rise to them, apparently by a perversion in the process by which cicatrisation takes place. A well-known instance is the formation of the so-called "amputation-neuromata," or "bulbous nerves," round or oval growths, the size of a bean or larger, which form on the divided extremities of the nerves in the stump left after an amputation. It has been supposed that neuromata may arise from a local neuritis, but this is doubtful.

The occurrence of new growths containing nerve-fibres may seem to be very remarkable. We must remember, however, how frequently they are connected with traumatic causes, *i. e.* take origin in cicatricial processes. In such processes the nerve-fibres present a remarkable power of growth. Ranvier has shown that from the end of each old fibre several new fibres grow, only one of which probably ultimately persists. Under apparently mechanical influences some of these fibres may twist about and even turn and grow upwards. It is thus not difficult to understand that a true neuroma may result from injury to a nerve; it is even surprising that this is not a more frequent result.

Almost any one of the cranial or spinal nerves may be the seat of a neuroma. They have been occasionally met with on the sympathetic.

SYMPTOMS.—These may be entirely absent, especially in the case of multiple (true) neuromata, which are sometimes discovered post mortem, when their existence has not been suspected. The most frequent symptom is pain referred to the distribution of the nerve, often acute, burning, or darting, and frequently intermittent. It may be increased by pressure on the tumour. In the case of the terminal neuromata (*tubercula dolorosa*, bulbous nerves) pain may be the only sensory symptom. When the nerve-fibres suffer, *paræsthesiæ*, numbness or formication, may be complained of, and ultimately sensation may be lessened or lost. Paralysis of the muscles supplied by the nerve is occasionally met with, the region paralysed corresponding to the distribution of the nerve on which the tumour is situated, except in the case of neuromata in the *cauda equina*, where the nerves may suffer from pressure, and paraplegia may result. In all cases the distribution of the symptoms depends necessarily on the nerve affected. A neuroma of the pneumogastric may cause grave cardiac disturbance.

More common motor symptoms are reflex spasms in adjacent or even distant muscles. For instance, in a case of "bulbous nerves" in the stump after amputation at the shoulder-joint, the muscles on that side of the neck were in constant clonic spasm. Occasionally epileptiform convulsions are produced, and have been known to cease when a

painful neuroma had been excised. The occurrence of these symptoms has little relation to the size of the tumour.

When the neuromata are in accessible situations they may be felt. Sometimes they give rise to visible tumours, and in multiple neuroma the course of the superficial nerves may be marked out by lines of bead-like swellings, as in Fig. 42. The cutaneous neuromata also cause visible swellings. The whole abdomen was covered with such nodules in the case recorded by Smith (Fig. 41). They are commonly not sensitive, although pre-existing pain may be increased by pressure. Pressure on the nerve above the tumour commonly lessens the pain.

Plexiform neuroma rarely causes other symptoms than the signs manifesting its local presence. The functions of the nerve-fibres involved are not usually interfered with. Once or twice anæsthesia of the skin has been observed.

Course.—The growth of neuromata varies greatly in rapidity. When symptoms occur they most commonly last for a long time, increasing in severity, and from the long-continued pain the patient may ultimately be worn out. In other cases the symptoms show little tendency to increase, and in rare instances they may lessen in severity and even disappear.

DIAGNOSIS.—The recognition of the existence of a neuroma depends on its superficial position, or on the production of symptoms of damage to the nerve. The latter, when existing alone, are equivocal, since they may be due to pressure on the nerve from an adjacent growth, or even to neuritis. The disease may be suspected if symptoms—pain, numbness, weakness—occur, limited to the distribution of a nerve-trunk, and no cause of external pressure can be discovered, and the long course of the symptoms makes it unlikely that they are due to neuritis. The diagnosis can, however, only be certainly made when the tumour can be felt.

The diagnosis of the kind of neuroma, whether “true” or “false,” *i. e.* composed of nerve-fibres or other tissue, is very difficult. Multiple neuromata are usually true, and of very slow growth; false neuromata are rarely multiple unless they are of infecting nature and rapid in development. The existence of idiocy, or other neuropathic indication, is in favour of the neural nature of the tumours. The lateral position of the growth on the nerve is in favour of its “false” character. The diagnosis of plexiform neuroma is only possible when the tortuous nodular cords can be felt.

PROGNOSIS.—This depends on the existence of symptoms. If these are absent, unless the tumour is of rapid growth, there is good reason to hope that they will not occur. In multiple (true) neuromata the prognosis is better than in isolated growths. If symptoms are present the prognosis is less favorable, and depends on the tendency which they exhibit to increase.

TREATMENT.—Medicines are useless except in the syphilitic forms of nerve-tumour. Extirpation is the only remedy. If the tumour is

lateral, and can be shelled out, the removal entails little risk. If, however, the growth infiltrates the substance of the nerve, the affected part must be excised, and the ends brought together. The risk of permanent loss of function in the nerve is of course great, and in deciding on such an operation, the urgency of the symptoms, and the importance of the function of the nerve, must be taken into consideration. After extirpation, the tendency of cicatricial processes in nerves to go on to the formation of neuromata (seen in bulbous nerves) renders the chance of relapse considerable. Electrolysis or caustics are inadmissible. When neuromata are multiple, surgical treatment is out of the question. For pain, sedatives must be employed. In the case of terminal neuromata, whether the cutaneous "tubercula dolorosa" or the amputation neuromata, excision is practicable and unattended with the risks attending interference with a nerve in its continuity. It is also usually successful. In amputation flaps, however, the chance of a relapse is considerable.

In plexiform neuroma, excision is undesirable, and no other treatment is of any use.

DISEASES OF SPECIAL NERVES.

The diseases of the cranial nerves may be most conveniently considered in connection with the diseases of the brain. Here, therefore, only the affections of the spinal nerves will be described, in so far as they present special features. The effect of paralysis of the individual muscles has been already described in detail, and need not be here repeated, except in general terms, or in so far as the association of palsies resulting from a nerve-lesion presents a particular character.

PHRENIC NERVE.—Impaired function of the phrenic nerve is commonly the result of disease of the spinal cord. The roots of the nerve may, however, be damaged by disease of the bones of the spine. The deep position of the nerve-trunk protects it from injury, but it is occasionally damaged in wounds of the neck, and in its course through the thorax it may be compressed by tumours, aneurisms, &c. Paralysis sometimes follows exposure to cold, and is ascribed to neuritis. It has been met with in lead-poisoning (Duchenne). In disease of the cord and bones, both nerves are usually paralysed; other causes commonly affect one only. The effect and symptom of paralysis is inaction of the diaphragm (see p. 22). If one nerve only is affected, the diaphragm does not descend on that side, but the movement of the other side lessens the resulting defect of movement, which can then be detected only by close observation. The loss of the action of the

diaphragm has little effect on the respiratory functions while the patient is at rest, but dyspnœa is readily produced by exertion; the breathing then becomes quick and the voice feeble. Any lung disease, such as an attack of bronchitis, is rendered far more serious by the diminished breathing power. When the diaphragm is paralysed, the movement of the thorax is often increased, and the expansion of the lower part may draw forwards the adjacent abdominal wall. This must not be mistaken for the effect of the descent of the diaphragm. Paralysis has to be distinguished from (1) abnormal nervous breathing. The diaphragm is used little in extraordinary breathing, which is chiefly by the upper part of the thorax, of the "superior costal type" as it is called. Hysterical and nervous patients will often breathe, for a time, only in this manner, even when at perfect rest, especially when they are under observation. Such breathing is no doubt facilitated in women by the fact that the diaphragm is habitually used by them less than by men. Repeated examination may be necessary to determine whether there is any real paralysis of the diaphragm in these cases. A single diaphragmatic inspiration settles the point. The patient's attention should be distracted, and she should not be aware of the object of the examination. (2) Inflammation of the diaphragm may arrest its movement, but is distinguished by the fact that it is usually secondary to adjacent inflammation, commonly of the peritoneum or the pleura. Characteristic pain, due to the inflammation of the serous membrane, is commonly present. (3) A primary and isolated degeneration of the muscular fibres of the diaphragm has been described by Callender and others as common after death, but it has not yet been proved to cause such inaction during life as might be confounded with paralysis. The distinction of paralysis, due to disease of the nerve-trunk, from disease of the spinal cord, depends on the fact that in the latter case other muscles always suffer; in the former the diaphragm usually suffers alone. In disease of the nerve-roots there are other indications of the position of the disease.

If there is reason to suspect neuritis, counter-irritation should be applied over the lower and inner part of the anterior triangle of the neck. The only other special point in treatment is the application of electricity. The nerve may be stimulated by pressing the rheophore deeply outside the lower part of the clavicular portion of the sternomastoid. The other pole may be placed at the epigastrium or over the corresponding half of the diaphragm. But the influence of electricity on paralysis of the diaphragm is not great.

NERVES OF THE UPPER LIMB.

The nerves of the arm and shoulder are derived from the five lower cervical and the first dorsal nerves. These interlace in the brachial plexus in such a complex manner that most of the nerves of the arm are derived from many spinal roots.

The nerve-roots form, by their union, three trunks, which we may indicate by Roman numerals. They are formed thus :—I, by the branch from the fourth, and the roots of the fifth and sixth cervical ; II, by the seventh ; and III, by the eighth cervical and the first dorsal. Each trunk divides into two parts, and the union of these divisions forms the three cords of the brachial plexus from which the nerves of the arm proceed. But before the primary trunks divide, certain nerves arise, the origin of which is thus less doubtful. The fifth and sixth cervical roots give origin directly to the posterior thoracic nerve for the serratus, and from the cord formed by their union springs the suprascapular nerve. The three cords of the plexus have the following relations :—The posterior is derived from all three primary trunks and gives rise to the subscapular nerve, the circumflex, and the musculo-spiral (or radial, as the whole nerve is sometimes termed). The upper or outer cord is derived from the two upper primary trunks, *i. e.* from the fourth, fifth, sixth, and seventh cervical roots, and from it proceed one anterior thoracic and the musculo-cutaneous nerves, together with the outer head of the median. The inner or lower cord is derived only from the lowest primary trunk, *i. e.* from the last cervical and first dorsal, and gives rise to the ulnar, the inner head of the median, the internal cutaneous, the intercosto-humeral, and to the second anterior thoracic nerves. It may be convenient to put these coarse anatomical relations in the form of a table.

<i>Nerves.</i>		<i>Primary Trunks.</i>		<i>Nerves.</i>	
Subscapular . Circumflex . Musculo-spiral }	Post. cord	I. 4, 5, and 6 C.	Upper cord	{ External ant. thoracic. Musculo-cutaneous. Outer head of } median.	
		III. (8 C.) (1 D.)	Lower cord	{ Inner head of } Ulnar. Internal cutaneous. Intercosto-humeral. Internal ant. thoracic.	

These anatomical facts, however, give us little help in tracing the relation of the nerves to the spinal roots. The investigations of Ferrier and Yeo,* who ascertained the movements produced by faradising the several spinal roots in the monkey, show us the way in which the movements, muscles, and nerves are represented in the spinal roots. Their results are therefore of great interest, although we are not justified in transferring the facts to man except in so far as they receive confirmation from human anatomy and pathology.† Subject to this reservation, the chief results are important and may be thus stated.‡

The roots to which the several nerves are thus traced are as follows : Subscapular, 6 and 7 C. ; circumflex, 4 and 5 C. ; musculo-spiral, 4, 5, 6, 7, 8 C. ; musculo-cutaneous, 4, 5 C. ; median, 5, 6, 7, 8 C. ; ulnar, 8 C., 1 D.

Still more important are their observations on the relation of various movements of the arm to the nerve-roots, and these are as follows :—

* 'Proc. Roy. Soc.,' March 21, 1881.

† In the case of the leg there are some important discrepancies between the results in the ape and the conditions that obtain in man. These will be noticed in their proper place.

‡ Ferrier has since stated that the relations he gave (followed in the text) were all one nerve too high ('Proc. Roy. Soc.,' 1883, vol. xxxv, p. 229), but this would make the innervation of the intrinsic muscles of the hand from the second dorsal nerve, which is certainly not the case in man.

Cervical 4.—Elevation and retraction of the arm, flexion and supination of the forearm; by the rhomboids, supra- and infra- spinatus, biceps, brachialis and supinators.

Cervical 5.—Similar to the last, but without retraction of the arm, and with extension of the wrist and first phalanges; by the deltoid, serratus, flexors of elbow, extensors of wrist, and long extensors of fingers.

Cervical 6.—Adduction and retraction of the upper arm, extension and pronation of the forearm, flexion of the wrist; by contraction of the pectoralis, latissimus dorsi, triceps, flexors of wrist, pronators.

Cervical 7.—Adduction and rotation inwards at the shoulder-joint, flexion of the wrist, and of the fingers at the second phalanx; by the teres major, latissimus dorsi, subscapularis, triceps, and long flexors of the fingers.

Cervical 8.—Flexion of fingers and thumb so as to close the fist; flexion of wrist towards the ulnar side, pronation of forearm, extension of elbow; by the intrinsic muscles of the hand, the long flexors of the fingers and thumb, the flexors of the wrist, and the triceps.

Dorsal 1.—Adduction of the thumb, flexion of the fingers at the metacarpophalangeal joints; by the interossei, &c.

Thus most movements are related to many spinal roots. The most important relations are these: that of the deltoid, rhomboids, supra- and infra- spinatus, flexors of the elbow, and supinators, to the fourth and fifth cervical; that of the adductors of the arm and extensors of the elbow to the sixth and seventh nerves; pronation to the sixth and eighth; extension of the wrist to the fifth; flexion to the eighth; extension of the first phalanx to the fifth; flexion of the fingers to the seventh and eighth, and the action of the intrinsic muscles of the hand to the first dorsal. These facts will probably, in the future, find important practical applications.

Paralysis of the nerves of the upper limb may be due to disease of the nerve-roots, of the plexus, or of the trunks that arise from these. It is convenient to consider, first, the diseases of the nerve-trunks, and afterwards those of the nerve-roots, and of the plexus. From the roots themselves only one important nerve-trunk is derived: the posterior thoracic nerve. The others spring from the plexus.

The morbid influences that affect the nerves of the arm are very varied. The brachial plexus passes close to the shoulder-joint, and hence dislocation often damages the nerves, sometimes one only, sometimes several, and occasionally all the trunks. In fractures of the bones, the deeper nerves adjacent may be torn at the time of fracture, or may be afterwards compressed by the callus that is formed, or by bandages that are applied. The course of certain nerves is, in some parts of the arm, especially apt to be the seat of wounds of various kinds, while that of others exposes them to pressure against the unyielding bone. Even slight pressure on these nerves is effective when prolonged, as it often is when warning sensations are unperceived in consequence of sleep. Hence, there is a group of "sleep-palsies" of the arm, chiefly of the musculo-spiral and median nerves.

Primary neuritis may involve any nerve, or the brachial plexus itself, but is not common. Secondary neuritis, set up by injury or by adjacent inflammation, is less rare; it sometimes extends upwards,

and, reaching the plexus, may spread to other nerves. Neuromata occasionally cause paralysis, but are also uncommon. Various morbid processes near the spine and in the neck may cause symptoms in the arms by involving the nerve-roots that enter the plexus.

Posterior Thoracic Nerve—Nerve to the Serratus.—In consequence of its position and long course, the nerve to the serratus often suffers. After being formed in the substance of the scalenus medius muscle, by the union of branches of the fifth and sixth cervical nerves, it passes behind the brachial plexus and along the side of the chest to the lower border of its muscle. It is in the neck that the nerve is most often injured. It may be damaged there by direct pressure—as in carrying on the shoulder a heavy sharp-cornered object—or by severe muscular efforts, such as carrying a weight that does not press into the neck, wielding a heavy hammer, or long-continued exertion with the raised arm, as, for instance, in whitewashing a ceiling. In such cases it is not improbable that the nerve is damaged by the forcible or prolonged contraction of the muscle through which it passes. Falls and blows on the neck and shoulder are occasional causes; there is usually much bruising of the adjacent parts. Punctured and gunshot wounds also occasionally involve the nerve. The least frequent cause is exposure to cold, such as a draught on the neck, or sleeping on damp earth. I have once known it to follow parturition, developing four days afterwards, doubtless from neuritis excited either by the muscular exertion, or by exposure to cold. The serratus suffers also in central disease, chiefly in progressive muscular atrophy and infantile paralysis; but in these it is always associated with palsy of other muscles. Isolated paralysis of the serratus is nine times as frequent in men as in women. It occurs chiefly in muscular workers, and during the active period of life, twenty-five to forty. The immediate causes sufficiently explain this relation to age and sex, and they also account for another fact—that it is far more frequent on the right side than on the left. It is sometimes bilateral, but the two nerves are never damaged at the same time. One man was affected on the right side after carrying heavy beams on the shoulder; he then carried them on the left shoulder, and the left serratus became paralysed.

Severe neuralgic pains in the neck and about the shoulder commonly precede and accompany the onset of the affection. The symptoms of the paralysis of the serratus have been already described (p. 24). It is readily recognised by the recession of the posterior edge of the scapula from the thorax when the arm is put forwards (Fig. 8, p. 25). In severe cases the muscle is found to have lost faradaic irritability, although it may still contract to the voltaic current. The course of a severe case is always tedious. Months may pass before improvement is manifested, and the paralysis is sometimes permanent. In treatment it is desirable to maintain the nutrition of the muscle, as far as possible, by electrical stimulation, and its superficial situation renders

this easy. If counter-irritation is desirable, it should be applied over the position of the scalenus, since the lesion of the nerve is commonly at this place. Severe exertion with the arm should be avoided; an influence capable of inducing the palsy must seriously interfere with the recovery of an injured nerve. When practicable it is desirable that the elbow should be carried in a sling, and this should be of such a length as slightly to raise the shoulder.

The *Suprascapular nerve* arises from the trunk formed by the union of the sixth, fifth, and a branch of the fourth cervical nerves, but its fibres come from the fifth and partly from the fourth. It is not often damaged alone, but is sometimes injured in dislocation of the humerus, and by falls on the shoulder, usually together with the circumflex. In some of these cases the injury may be to the upper part of the plexus (see "Combined Palsies"). The result of disease of this nerve is palsy of the supra- and infra- spinatus (p. 26). The latter is the more important, and causes a loss of the rotation outwards of the humerus, interfering with many movements, and, among others, with the movement of the pen along the line in writing. Undue work is thrown on the deltoid, which becomes fatigued in many actions, such as sewing (Duchenne). The paralysis of the supraspinatus is unimportant unless there is also palsy of the deltoid. It can, in very slight degree, supplement the deltoid, and if paralysed with the deltoid the head of the humerus falls more than it does when the deltoid is paralysed alone.

The *Circumflex nerve*, although arising from the posterior cord of the plexus, seems to derive its fibres from the same source as the suprascapular, the fourth and fifth cervical nerves. It supplies the deltoid and teres minor, and the skin over the deltoid. The course of the nerve renders it very liable to injury from dislocations and falls on the shoulder, and from the pressure of a crutch. Simple neuritis and so-called "rheumatic palsy" are rare, but the nerve has been affected in some acute diseases, as smallpox, possibly from pressure, and in rheumatic fever, perhaps by inflammation extending from the joint. It suffers, with other nerves, from disease of the upper part of the brachial plexus.

The chief symptom is paralysis of the deltoid (p. 25), which abolishes almost all power of raising the arm, a very trifling power of abduction by the supraspinatus alone remaining. The slight nerve-supply that the fore part of the deltoid receives from one of the anterior thoracic nerves is insufficient to mitigate the effect of the paralysis of the circumflex, although it may maintain slight power of voluntary contraction in the anterior fibres, and this sometimes gives rise to an erroneous impression that the paralysis of the circumflex nerve is incomplete. The effect of the palsy of the teres minor is unimportant. The wasting of the deltoid causes a change in the shape of the shoulder. Sensation may be lost in the skin over the lower part of the muscle, and the loss sometimes extends on to

the shoulder (Fig. 46). In some cases there is no anæsthesia, even when the muscle is wholly paralysed, we have seen that this is often the case in nerve-lesions (p. 51). Adhesions are apt to form in the shoulder-joint, in part probably the result of trophic changes, since the circumflex supplies the articulation as well as the chief muscle that moves it. The diagnosis of the paralysis of the circumflex nerve is easy. The only condition readily mistaken for it is ankylosis of the shoulder-joint in a stout individual, in whom the state of nutrition of the muscle is not readily perceived. The risk of error is increased by the fact that arthritis and paralysis may result from the same cause—a fall on to the shoulder. Passive movement at once solves the problem. The scapula moves with the arm in ankylosis, and not in palsy.



FIG. 46.—Disease of the circumflex nerve from pressure-neuritis during acute illness; wasting of deltoid. The dotted line indicates the area of cutaneous anæsthesia.

The *Musculo-cutaneous nerve* supplies the chief flexors of the elbow and the skin over the radial side of the forearm. It is scarcely ever paralysed alone, but often suffers, with other nerves, in disease of the brachial plexus. The symptoms correspond to its function; there is paralysis of the biceps and brachialis (see p. 27), the effect of which is especially conspicuous when the arm is supinated and the supinator longus cannot flex the elbow. There may also be anæsthesia of the radial side of the forearm, front and back.

The *Musculo-spiral nerve* is more frequently paralysed alone than any other nerve of the arm. Arising from the posterior cord of the brachial plexus, it seems to derive its motor fibres from all the nerve-roots that enter the plexus except the first dorsal. It supplies the triceps, all the muscles on the back of the forearm, the extensors of the wrist and fingers, both the supinators, also the skin on the radial side of the back of the hand, the back of the thumb, index finger, and half the middle finger. It is thus the extensor nerve of the arm, and has a more complete relation to a single function than is common among the nerves of the limbs; a fact that has given rise to diagnostic error, since a functional palsy suggests a central cause. The frequency with which this nerve suffers is due to its course. As it leaves the brachial plexus to wind round the bone, its position exposes it to injury in dislocation or from the pressure of a crutch; the most common form of "crutch palsy" is paralysis of this nerve. Lying as it does close to the humerus, it is readily torn by fracture, or compressed by callus, and it suffers gravely from even temporary pressure against the hard bone. Such pressure is often exerted during sleep. The nerve may be paralysed by the pressure to which it is exposed when the body, with the arm beneath it, rests on the ground

or on a hard bed. Thus, a man slept all night on a bench, lying on his right side with his arm beneath him, and woke in the morning with this nerve paralysed. Curiously, a precisely similar event had occurred to him three years before. In other cases the pressure on the nerve is that of a hard and sharp object over which the arm is placed during sleep, as the edge of a chair or the side of a couch. The continental custom of tying together, behind the body, the arms of a prisoner, often causes paralysis of this nerve, sometimes on both sides. In Russia it is not uncommon in infants, from the popular practice of binding the arms to the body and then laying the child to sleep on its side. I have twice seen paralysis from a violent contraction of the triceps, once during the act of pulling on a tight pair of boots, and once from throwing a stone with violence. In each the nerve was at once completely paralysed; and in the second, in which the palsy was severe, a bruised appearance was observed over the lower part of the triceps. Neuritis due to cold is often assumed to be a cause and is possibly sometimes effective, but the exposure to cold has usually been during sleep, and, as Panas and others have maintained, it is probable that the paralysis was due to compression rather than to cold. The same influence may also have been operative in cases in which the nerve has been paralysed during an acute disease. Although, in a case of paralysis during typhus, described by Bernhardt, neuritis was found after death, this was at the spot at which pressure is usually effective, and the inflammation was probably thus excited. The muscles involved in lead palsy are some of those supplied by the musculo-spiral, but we do not know that the poison acts on the nerve itself.

The symptoms of disease of the musculo-spiral nerve are paralysis of the extensors of the elbow and of the wrist, the long extensors of the fingers and thumb, and the supinators (see pp. 26-28). All these are paralysed by a complete lesion of the nerve near the brachial plexus. When the lesion is near the middle of the humerus, as it is in most cases of compression, the triceps generally escapes, but not always. The supinator longus is usually paralysed, but it escapes if the lesion is below the origin of its branch, and may also escape if the injury to the nerve is incomplete. In sleep-palsy it usually suffers, but I have seen it unaffected in a case in which the ext. carpi radialis was also but little affected, and Bernhardt has noted the escape of the supinator in an otherwise complete paralysis from dislocation of the humerus. The extensor palsy causes characteristic wrist-drop, and loss of the power of extending the first phalanges of the fingers and the thumb (see p. 28). There is sometimes a gradation of palsy from the first finger, in which it is least, to the fourth, in which it is greatest (Fig. 47). The same gradation is seen in lead palsy. It is not easy to explain. The action of the flexors is feeble, from the loss of support; in most cases of complete palsy I have found the power of flexion reduced from this cause to one third of the normal. The loss of the

power of supination is a grave inconvenience. If an object is grasped firmly the arm becomes pronated. The patient tries to compensate for



FIG. 47.—Paralysis of the musculo-spiral nerve; maximum extension of wrist and fingers. The extension of the fingers progressively diminishes from the first to the fourth. (From a photograph.)

the loss by putting the elbow against the side and rotating the humerus. The pronators may ultimately become shortened. The over-flexion of the carpus, and its deficient support by the extensor tendons, leads to a prominence of synovial sacs, and perhaps of the bones, at the back of the carpus (Fig. 48). In severe cases the muscles waste, and the maximum circumference of the limb below the elbow may be a quarter or half an inch less than on the other side. The electrical reactions depend on the severity of the lesion of the



FIG. 48.—Prominence at back of hand from paralysis of the extensors. The patient was suffering from wrist-drop due to silver-poisoning.

nerve; commonly there is well-marked degenerative reaction. The affection of sensibility in the area supplied by the nerve is very variable. The skin of the upper arm rarely loses feeling; in the hand sensation may be normal, although the muscular paralysis is complete. There may be subjective "tingling" in the part although there is no loss of sensibility. The diagnosis is easy in most cases. The affection is distinguished from lead palsy by its common limitation to one arm and by the affection of the supinator, but these distinctions, although strongly suggestive, are not quite absolute. The palsy of the nerve, moreover, is usually sudden in onset; that from lead is gradual. These characters, and the commonly obtrusive cause, always suffice for the diagnosis. In bilateral wrist-drop, disease of the nerve would not be thought of, unless it followed a cause acting on both sides. It must be remembered, however, that this nerve is one most frequently affected in multiple neuritis; in this disease both arms suffer, the legs

are also often involved, pains are conspicuous, and the palsy is rarely limited to a single nerve-region. The prognosis in disease of the musculo-spiral nerve depends on the severity of the lesion, as indicated by the electrical reaction. When there is evidence of nerve-degeneration the paralysis usually lasts for some months. Recovery ultimately occurs in almost all cases. The treatment is that for neuritis already described, but attention should be paid to the posture of the limb, so as to avoid tension on the affected part of the nerve. For this reason, and also because strong contraction of the triceps may further injure the nerve, extension of the elbow should be avoided in cases of lesion of the nerve as it winds round the humerus.

The *Median nerve* supplies the pronators, the radial flexor of the wrist, the flexors of the fingers (except the ulnar half of the deep flexor), the muscles that abduct and flex the thumb, and the two radial lumbricales. Its motor fibres seem to have an extensive origin from all the cervical roots that enter the brachial plexus. It subserves sensation on the radial side of the palm, on the front of the thumb, of the first two fingers, and of half the third finger, and also, at least in many persons, on the back of the last phalanx of the index and middle finger; sometimes also on the adjacent part of the back of the ring finger and the back of the last phalanx of the thumb. Isolated palsy of this nerve is not frequent, and generally results from wounds of the forearm, or fractures of the forearm bones, rarely from injuries in the upper arm. The nerve is occasionally the seat of neuritis. I have known it to be paralysed at the wrist a few hours after a severe sprain of this joint. Webber has recorded a case in which it seemed to be injured by a violent contraction of the pronator teres. When there is complete damage to the nerve, pronation is impossible beyond the mid-position, to which the supinator longus can bring the forearm, and an attempt is made to supplement this by rotating the humerus inwards, and separating the elbow from the side. The wrist can only be flexed with a strong inclination towards the ulnar side. The thumb is in persistent extension and adduction (like the thumb of the ape, Figs. 20 and 21, p. 32), and cannot be opposed to the tips of the fingers. The power of flexing the second phalanges on the first is lost, and also that of flexing the distal phalanx of the first and second finger, but this phalanx can still be flexed in the third and fourth fingers, by the ulnar half of the flexor profundus (see p. 29). Interosseal flexion of the first phalanx is still possible, and the unopposed extensor action of the interossei on the middle and distal phalanges tends to cause a subluxation of the articulations concerned. The wasting of the thenar muscles is usually conspicuous. The condition of the hand resulting from these palsies is very characteristic. The sensory loss is variable; it may be absolute or absent. If there is anæsthesia on the palmar surface it will often be found also on the dorsal aspect of the extremities of the first two fingers. Lesions of the median nerve, if rare, are apt to be severe, and their course correspondingly prolonged.

The *Ulnar nerve* comes, through the inner cord of the plexus, from the last cervical and first dorsal roots, and its origin from the lowest part of the cervical enlargement gives it an important relation since it is the first, of all the brachial nerves, to be affected by disease that ascends from the dorsal to the cervical region of the spinal cord. The nerve supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the interossei, some of the lumbricales, the adductor and inner head of the short flexor of the thumb. Its sensory area is the ulnar side of the hand, back and front, more extensive on the back of the hand (two fingers and a half) than on the front (one finger and a half). The course of the nerve, superficial behind the elbow and at the wrist, renders it liable to separate injury. It is often implicated in wounds of the arm, and injured in operations about the elbow-joint, by dislocations of the shoulder and elbow, and by fractures of the bones of the forearm. It is occasionally the seat of neuritis, and, like the musculo-spiral nerve, it is sometimes paralysed by pressure. It is probable that pressure often acts by setting up neuritis. At the elbow, the projecting bones preserve the nerve from direct pressure; although Duchenne describes paralysis in men who, while at work, rest the bent elbow on a hard substance, this mechanism is certainly rare. It is much more common for the nerve to suffer in long-continued flexion of the elbow, without external pressure; and from this cause the nerve is sometimes paralysed during sleep. I have three times seen a sleep-palsy of the ulnar nerve. The tension may set up neuritis, and is especially effective if the resistance of the tissues to morbid influences is lessened by general ill-health. Many persons must have noticed that if, when out of health, they sleep with the elbow bent, they wake with tingling, and even loss of feeling, in the region supplied by the ulnar nerve, although this may not happen when they are in good health. If the ill-health is profound, as in an acute illness, local neuritis of considerable intensity may be set up. Thus a lady, a few days after childbirth, who was prostrated by a long, exhausting labour, noticed tingling in the side of the hand, and, whenever she bent her elbow, a sensation "as if she had knocked her funny-bone." Paralysis of the muscles supplied by the ulnar nerve followed in a few hours, and in a fortnight there was distinct wasting. When I saw her, six months later, the ulnar nerve behind the elbow, was distinctly thickened. She had had a similar, but more transient, attack after a previous confinement. Another patient had a similar palsy during the course of typhoid fever. Bernhardt has described a case of palsy of both ulnar nerves during the course of typhoid. In such conditions, not only is the tissue-health lowered, but warning sensations are often unperceived.

The importance of the interosseal flexion of the fingers for many actions, such as writing, renders palsy of the ulnar nerve peculiarly disabling. Several illustrations of the symptoms have been given in connection with palsy of the interossei (p. 30). In flexion of the wrist, the

hand deviates towards the radial side, and persistent distortion may take place. Adduction of the thumb is lost, and so are most movements of the little finger. The fingers cannot be flexed at the first or extended at the other joints, but the loss is slighter in the first two fingers than in the others, because their lumbricales are supplied by the median nerve. In time the opponents of the interossei, by their contracture, lead to over-extension of the first phalanges and flexion of the others; the "claw-like hand" is produced (see p. 30), but this is less complete than in cases of progressive muscular atrophy, on account of the escape of the first two lumbricales. When the palsy is imperfect it may be possible to extend the second and third phalanges, if the first can be over-extended, and an advantage thus given to the interossei, by the lengthening of the course of their tendons, just as in partial paralysis of the long extensors they may be able to extend the digits if the wrist is flexed, but not if it is extended.

The loss of sensation, as in the case of the other nerves, varies much both according to, and irrespective of, the degree of lesion of the nerve. Subjective sensations are common, with and without anæsthesia, and those who have had neuritis are sometimes unable to bend the elbow for more than a few minutes without feeling tingling in the area supplied by the nerve.

The diagnosis of disease of the ulnar nerve is easy. A difficulty is occasionally caused by the circumstance already mentioned, that disease at the lowest part of the cervical enlargement may cause symptoms limited to the region of this nerve, but a knowledge of the fact is usually sufficient to prevent an error.

COMBINED PALSIES OF THE NERVES OF THE ARM.—Paralysis in the region of two or more of the nerves of the arm is very common, and results from many causes. It may be due to disease of the spinal cord, or to disease of the nerve-roots within the spinal canal, but with these we are not now concerned. The most frequent causes of such combined palsy outside the spinal canal are the following:—(1) Morbid processes in the neck, affecting the nerve-roots outside the spine or the upper part of the brachial plexus. (2) Diseases of the plexus itself, neuritis, and especially dislocations of the shoulder. (3) Fractures of the bones of the forearm. (4) Ascending neuritis, "neuritis migrans," which, commencing in a single nerve, may ascend it to the plexus, and there spread to the fibres of other nerve-trunks.

We may consider first the cause last named. An injury of a single nerve is sometimes followed by an extension of the motor and sensory symptoms to the regions supplied by other nerves. This can only be explained by an ascending neuritis reaching the junction of the nerves at the brachial plexus, and there spreading, often in what seems a random manner. Thus a lady cut her wrist so as to leave a scar an inch long, over the position, and in the direction, of the median nerve.

The injury caused paralysis and wasting of all the muscles in the hand supplied by this nerve. This palsy was followed by a gradual loss of power, with diminished faradaic irritability, in the long extensor of the fingers, the ulnar flexor of the wrist, and also by defect of sensation in the skin supplied by the ulnar nerve. Hence the neuritis must have ascended to the plexus, and there have spread along each root of the median nerve. Again, a woman cut her right hand with a broken stone bottle, along the hypothenar eminence. There weeks afterwards the muscles of the hand supplied by the ulnar nerve gradually became paralysed, with wasting and loss of irritability, and in the area of the skin supplied by the nerve there was first tingling and then diminished sensibility. Burning pain spread up the front of the forearm, and inner side of the upper arm, to the axilla; all the forearm muscles lost power, the flexor carpi ulnaris, and the flexor sublimis digitorum in greatest degree; the tingling and lessened sensibility spread to the fingers supplied by the median nerve, and the abductor pollicis wasted. In this case also there must have been an ascending neuritis of the ulnar, spreading to the median at the brachial plexus.

The brachial plexus is occasionally the seat of a primary neuritis, sometimes apparently rheumatic in origin, sometimes developing without any distinct exciting cause. The symptom of such neuritis is pain, acute or burning in character, in the position of the plexus, extending along the course of some of the nerve-trunks, and accompanied by various indications of organic damage to the nerves that arise from the plexus. These cases are always most tedious in their course. Fortunately they are rare. A far more common cause of damage to the plexus is dislocation of the shoulder, especially subcoracoid dislocation, in which the displaced head of the humerus necessarily compresses the nerves. The extent of injury varies greatly; only one nerve may suffer, or not one may escape. Every muscle of the arm may be paralysed, from the deltoid downwards. In most cases the injury is severe in degree; there is rapid wasting of the muscles, with the reaction of degeneration, and there is a great tendency to the occurrence of trophic changes in the skin. It was in a case of this character that the forearm and hand were covered with blisters from the application of water that seemed merely warm to a healthy hand (p. 52).

Fracture of the humerus may damage both the musculo-spiral and ulnar nerves, rarely the median. Fracture of the bones of the forearm often injures both the median and ulnar nerves.

Injuries to the neck sometimes cause a partial paralysis of the arm of peculiar distribution, the special characters and significance of which were first pointed out by Erb.* A similar paralysis sometimes comes on apart from injury. The muscles affected are the deltoid,

* Heidelberg Society, 1874, 'Ziemssen's Handbuch,' 1874, Bd. xii, pt. 2, p. 509; see also Bernhardt, 'Zeitsch. f. kl. Med.,' 1882, Bd. iv, p. 415.

often the supraspinatus and infraspinatus, the biceps and brachialis

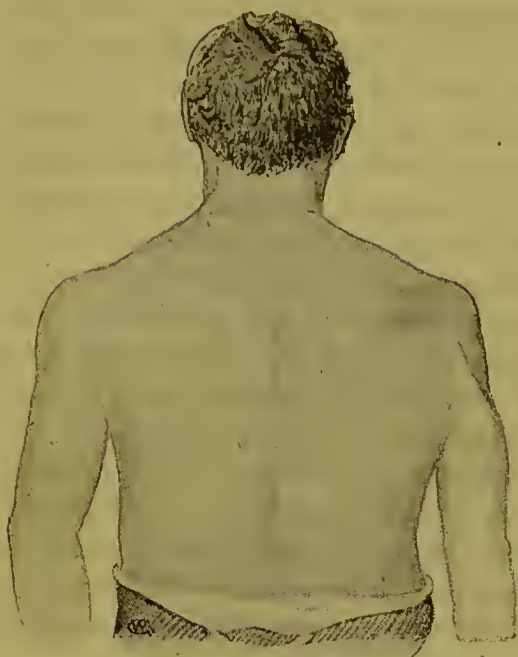


FIG. 49.—Combined palsy of deltoid, supraspinatus, and infraspinatus, from a fall on the shoulder.



FIG. 50.—Left hand of a patient suffering from a growth beside the lower cervical spine compressing the nerve-roots. There was anaesthesia of all parts supplied by the brachial and cervical plexus. The arm was adducted, the elbow flexed, the hand in the posture shown, flexion of first, extension of second, strong flexion of last phalanges, the first phalanx of the thumb over-extended, the second flexed. Rigidity extreme, and an attempt to overcome it caused great pain. There was also some contracture of the foot (equino-valgus).

* Erb referred the palsy to disease of the sixth nerve. The experiments of Ferrier and Yeo point to the fifth and fourth roots as those innervating these muscles, but his subsequent correction indicates the fifth and sixth (see p. 67).

anticus, and the supinators. Erb found that there is one spot between the scapuli, corresponding to the sixth cervical nerve, at which electrical stimulation puts all these muscles in action. Hoedemaker, who has described two cases of this palsy, finds the motor point in a line drawn from the sterno-clavicular articulation to the seventh cervical spine, 1.5 centimetres from the edge of the trapezius. The palsy is apparently dependent on disease of the roots of the fifth and sixth cervical nerves, and the fifth, it will be remembered, receives a twig from the fourth.* Besides injuries, this group of palsies may result from non-traumatic processes, probably from neuritis, and also from growths in this situation. Duchenne and Seligmüller have also described paralysis of similar distribution produced in infants during birth, either by pressure from the position of the arm, or by traction on the neck with the finger or hook. Most obstetrical cases slowly recover. Those due to injury in adult life are often severe; the symptoms continue for a long time, and may be permanent.

Morbid processes connected with the bones of the cervical

spine sometimes produce combined symptoms of irritation and palsy of the nerves of the arm, pain, hyperæsthesia, anæsthesia, paralysis of muscles, and extreme muscular contractures, often very irregular in distribution (such as are shown in Fig. 50). I have also twice seen purely sensory symptoms in the arm due to what was apparently syphilitic cellulitis about the vertebræ, causing deep-seated induration beside the spine, and quickly dispersed by iodide of potassium.

DIAGNOSIS.—The diagnosis of diseases of the nerves of the arm has been for the most part sufficiently considered in connection with the special nerves. It consists chiefly in an application of the facts there stated. One or two more general considerations deserve, however, a brief mention. Some diseases of the spinal cord are first manifested in the arm, by muscular palsy, wasting, or by anæsthesia. The risk of error is prevented, in most cases, by the absence of any correspondence between these symptoms and the functions of special nerves, by the absence of any indication of morbid processes in the neighbourhood of the nerves, and by the presence of other signs of disease of the spinal cord. It has been mentioned that disease in the lowest part of the cervical enlargement may be manifested only in the region of the ulnar nerve, the lowest in origin of all the brachial nerves. Such disease is, however, usually bilateral, and secondary to disease in the dorsal region of the cord, the indications of which have preceded the symptoms in the arm. A knowledge of these facts, and a careful consideration of the distribution and course of the symptoms, will rarely leave the observer in doubt.

A greater difficulty in diagnosis is presented by some cases of functional disorder. The arm is a common seat of neuralgia, and the diagnosis between neuralgia and neuritis is often one of great difficulty, a difficulty that is increased by the circumstance that slight disseminated neuritis may unquestionably result from a primary neuralgia, so that the nerve-trunks may become tender. The distinction rests on the variable and intermittent character of the initial pain, on the secondary character of the tenderness of the nerves, and on the absence of interference with the function of their fibres. A similar difficulty is presented by some cases of "occupation neurosis," of which "writers' cramp" is the most common form. Sensory symptoms, common in some degree in these affections, may predominate over the motor symptoms, and may consist of nerve pains and tenderness. The fact that the pain is at first excited only by one kind of muscular effort, the initial limitation of the disability, and the absence of any actual paralysis, motor or sensory, usually suffices for the diagnosis. These cases are considered more fully in the chapter on "Occupation Neuroses."

TREATMENT.—There is little in the treatment of the diseases of the nerves of the arm that requires special mention. The chief measure is the treatment of the cause of the paralysis. Any present source of

pressure must be removed as far as possible. The ends of a divided nerve must be sutured. It is remarkable how quickly this has sometimes been followed by the restoration of conducting power. The posture of the arm must, in all cases, be such as to avoid tension on an injured or inflamed nerve. The treatment of neuritis is that already described. Electrical stimulation of the muscles is of great importance in all cases of severe damage to the nerves. Even in old and stationary cases it sometimes is the means of starting some improvement.

NERVES OF THE LOWER LIMB.

Disease of the nerves is far less common in the lower than in the upper limb, with one important exception,—the primary disease of the sciatic nerve that goes by the name of “sciatica.” This affection is reserved for separate description in the next section.

The nerves of the leg are derived from the lumbar and sacral plexuses. The *lumbar plexus*, consisting of the first three and a half lumbar roots, supplies the skin of the lower part of the abdomen, of the front and sides of the thigh, of the inner side of the lower leg and foot. It supplies also many muscles—the cremaster, and those that flex and adduct the hip-joint, and those that extend the knee. Its branches for the leg are the obturator and anterior crural nerves. The *sacral plexus* consists of the fifth lumbar root and half the fourth (lumbo-sacral cord) and the first four sacral nerves, of which, however, only the upper three have to do with the leg. This plexus innervates the extensors and rotators of the hip, the flexors of the knee, and all the muscles that move the foot, together with the skin of the gluteal region, the back of the thigh, the outer side and back of the lower leg, and most of the foot. Its chief nerves are branches to the outward rotators of the hip, the gluteal nerve, and the small and great sciatic.

The results obtained by Ferrier and Yeo (see p. 67) on stimulating the roots of the lumbar and sacral plexuses in the monkey, may be thus summarised:

Lumbar I and II. Lower abdominal muscles (not cremaster).

III. Psoas and iliacus, sartorius, extensors of knee; (flexion of hip and extension of leg).

IV. Glutei, adductors, extensor cruris, peroneus longus; (extension of hip and knee, elevation of outer side of foot).

V. Glutei, hamstrings, and all the muscles in front and back of lower leg; (rotation of thigh outwards, flexion and rotation inwards of leg; extension of foot with elevation of outer edge; flexion of distal phalanges of toes).

Sacral I. Hamstrings, calf muscles, long flexor of toes, intrinsic muscles of foot; (slight outward rotation of thigh, flexion of knee, extension of foot, adduction of great toe, flexion of first phalanges of all toes and of both phalanges of great toe).

II. Intrinsic muscles of foot; (“interosseal” flexion of toes, similar to the last).

These results cannot be simply applied to man. For instance, it is certain

that in man, one flexor of the hip, the psoas, and the cremaster, are largely innervated from the second lumbar, but no indication of the action of either of these could be observed on stimulating this root in the monkey.

The *Lumbar plexus* itself is sometimes damaged by growths in the abdomen (especially by those that spring from the lumbar glands) and by psoas abscess, and the nerve-roots may suffer in disease of the bones of the vertebræ. The plexus may also suffer when inflammation ascends the lumbo-sacral cord from the sacral plexus, and it is occasionally the seat of spontaneous neuritis. The *obturator nerve* is rarely affected alone; it has been sometimes damaged in the course of parturition. The *anterior crural nerve* has suffered from the same cause, and is sometimes injured by wounds of the groin or thigh, or by dislocation of the hip-joint.

Of the interference with movement that may result, the paralysis of the flexors of the hip depends on the position of the disease. This paralysis is total only when the lumbar plexus is damaged. Disease of the anterior crural nerve, within the abdomen, does not affect the psoas, but may paralyse the iliacus, and so weaken, without abolishing, the power of flexion. The chief symptom of disease of this nerve is the loss of power in the extensors of the knee, and the wasting of these muscles, together with loss of the knee-jerk which results from the interruption of the reflex arc. The effect of these palsies on movement is very serious (see p. 33). Anæsthesia involves the whole of the thigh (except a strip along the middle of the back), the inner side of the leg and foot, and the adjacent sides of the first and second toes. In many cases the paralysis is incomplete, and the symptoms present corresponding variation. Irritation of the nerve may cause severe pain in the region supplied by it. This is sometimes an early symptom of a growth near the spine. The pain in neuritis of the plexus may extend along the course of the ilio-hypogastric, ilio-inguinal, and genito-crural nerves, to the lower part of the abdomen and groin.

Paralysis of the obturator nerve causes a loss of the power of adduction of the thigh, so that the patient cannot put one leg across the other. Rotation of the thigh outwards is interfered with, but in slighter degree than adduction. The effect of these palsies (described on p. 33) is far less serious than is the disability which results from disease of the anterior crural.

The *Superior gluteal nerve* occupies an intermediate position between the two plexuses, arising, as it does, from the lumbo-sacral cord, which descends from the fourth and fifth lumbar roots. Its disease, which is very rare in isolated form, causes paralysis of the gluteus medius and minimus, with a loss of abduction and circumduction of the thigh (see p. 33).

The *Sacral plexus* is sometimes damaged by growths in the pelvis, by pelvic inflammation of various kinds, and by compression during parturition. It is also often the seat of neuritis, which, however, less frequently begins in the plexus, than ascends to it from the sciatic

nerve. Apart from spontaneous neuritis, which will be separately described, the sciatic nerve outside the pelvis is occasionally injured by wounds, rarely by dislocations of the hip, often by disease of the bone, and by adjacent morbid growths. It is also a relatively frequent seat of neuroma. Of the terminal branches, the external popliteal, by its superficial course, and proximity to the hard fibula, suffers from traumatic lesions of various kinds; it is also prone to spontaneous neuritis. This nerve is homologous with the musculo-spiral nerve of the arm, and presents an analogous liability to disease. The posterior tibial nerve is more secure in its deeper course, but may be damaged by fracture of the bones.

The symptoms of palsy of the sciatic nerve vary much in their character, according to the position of the disease. The *small sciatic* is implicated only when the mischief involves the pelvic plexus, and it scarcely ever suffers alone. The effect is palsy of the gluteus maximus, which interferes with rising from a seat more than with walking (see p. 33). There is also anæsthesia of the skin in the middle third of the back of the thigh, and in the upper half of the calf. A lesion of the sciatic nerve, near the sciatic notch, paralyses the flexors of the leg (which are also extensors of the hip; see p. 34), and all the muscles below the knee. If the lesion is below the upper third of the thigh, the flexors of the leg escape. The anæsthesia that results from a lesion of the nerve below the origin of the small sciatic, involves the outer half of the leg, the greater part of the dorsum of the foot, and all the sole.

The symptoms of disease of the branches of the sciatic are as follows:—

That of the *external popliteal nerve* causes paralysis of the tibialis anticus, long extensor of the toes, peronei, and extensor brevis digitorum. The effect of this is a loss of all power of flexing the ankle and of extending the first phalanx of the toes (see p. 35). The foot cannot be raised from the ground in walking, and talipes equinus ultimately results (Fig. 24, p. 36), which may be attended with persistent flexion of the first phalanges of the toes from contracture of the unopposed interossei. There is also anæsthesia on the outer half of the front of the leg, and on the dorsum of the foot.

Disease of the *internal popliteal nerve* paralyses the popliteus, calf muscles, tibialis posticus, and long flexors of the toes, as well as the muscles of the sole. In addition to the disability which characterises paralysis of the plantar muscles, there is loss of the inward rotation of the flexed leg, if the disease is so high as to involve the branch to the popliteus, and there is also loss of the power of extending the ankle-joint. Talipes calcaneus results (Fig. 23, p. 35). The sensory loss is anæsthesia on the outer part of the back of the leg in the lower half, and on the sole.

The *plantar nerves* rarely suffer alone. A lesion of the *internal nerve* causes anæsthesia on the inner part of the sole, and plantar surface of

the three inner toes and half the fourth, together with paralysis of the short flexor of the toes, the plantar muscles of the great toe (except the adductor), and of the two inner lumbricales.

Disease of the *external nerve* produces anæsthesia of the skin on the outer half of the sole, the little toe and half the fourth, paralysis of the flexor accessorius, the muscles of the little toe, all the interossei, the two outer lumbricales, and the adductor of the great toe. The effect of this palsy (see p. 37) is serious, since the toes cannot take their proper share in propelling the body forward in walking, and they gradually become flexed at the last two joints and extended at the others, from the contracture of the opponents of the interossei,—a position of the toes that causes serious inconvenience in walking.

DIAGNOSIS.—The diagnosis of diseases of the nerves of the leg is determined by the same general principles as those that have been mentioned as applicable to the nerves in general, and to the nerves of the arm in particular. The limitation of the symptoms to the functional areas of individual nerve-trunks, the evidence afforded by nutrition and irritability that the muscles are separated from the spinal cord, the implication of the sensory functions, and often the tenderness of the affected nerves, indicate, in most cases, the seat of the disease. To these signs, are often added other indications of a local cause, corresponding, in its incidence, with the nerve to which the symptoms point.

The relation of nerve-trunks to nerve-roots, although by no means simple, is certainly less complex in the case of the nerves of the leg than in those of the arm. This is especially the case in the lumbar plexus, and it leads to an occasional difficulty in diagnosis. Pressure on the spinal cord, for instance, at the level of the origin of the fourth lumbar roots, may cause symptoms identical with those of a partial lesion of the anterior crural nerve. Paralysis of the extensors of the knee, loss of the knee-jerk, anæsthesia in the front of the thigh, were present from this cause in a case of meningeal gumma. But other evidence of a spinal lesion is rarely absent under such circumstances, and, in this patient, a foot-clonus, due to the pressure on the pyramidal fibres, left no doubt as to the situation of the disease. Another difficulty arises from the long course of the nerve-roots in the cauda equina, disease of which may simulate that of the nerves of the leg. But the symptoms are commonly bilateral in consequence of the proximity of the nerve-roots of the two sides. In all cases in which symptoms are bilateral (unless there is evidence of a disease known to cause symmetrical lesions, such as multiple neuritis), the suggestion is that the disease is situated where the motor or sensory paths of each side are so near that they can be affected by a single lesion, *i. e.* that the disease is within the spinal canal. But here, as in other cases, we cannot reverse our diagnostic rules. Disease of the spinal cord does not always cause bilateral symptoms. A limited lesion of

one anterior cornu may be so placed as to paralyse the muscles supplied by a single nerve, and a doubt may be felt as to the central or peripheral origin of such palsy. The muscles supplied by the anterior crural nerve, and the muscles in the front of the lower leg supplied by the external popliteal, are those of which the central palsy sometimes leads to doubt. But the mode of onset, the presence or absence of sensory symptoms, the rarity of acute spinal palsy except in childhood, and of nerve-lesions except in adult life, the wider initial prevalence of the palsy in acute, and its later extension in chronic, cornual disease,—these suffice as a rule to remove any doubt that may at first be felt.

It is important to remember that the pressure of a growth may cause either a chronic or an acute affection of the nerves. The chronic symptoms result from pressure; the acute from a neuritis set up by the pressure and irritation. Thus neuritis, while it does not suggest, does not exclude, a cause outside the nerve.

TREATMENT.—The treatment of diseases of the nerves of the leg must depend, in each case, on the nature of the lesion and the character of the symptoms. It does not differ from that of disease of the nerves of the arm, and its general principles have been already stated. More care, perhaps, is needed to avoid increasing present mischief, or inviting a relapse, by exposure to cold, or by fatiguing exertion. More care is also needed to obviate the tendency to secondary contractures in the case of palsies of long duration, and in those attended by pain, in which the patient seeks ease in postures to which the muscles only too readily adapt themselves. The contraction of the hamstrings, from constant flexion of the knees, is that which occurs most readily, and is most serious when the patient recovers from the palsy, or loses the pain, which led to the adoption of the position. The contracture of the calf muscles, which occurs when the flexors of the ankle are paralysed, also constitutes a serious obstacle to walking after recovery. A little timely care, in preventing these contractures, will often save a vast amount of later trouble. That of the calf muscles, however, which is due to the extension produced by the weight of the foot, as the patient lies, cannot always be altogether prevented.

SCIATICA.

As the word *sciatica* is commonly used, it is a general designation for all affections of which the chief symptom is pain in the region of the sciatic nerve. It is thus often applied to the pain which results from pressure on the nerve in the pelvis, as by a tumour. In a stricter use of the word, however, it is applied to painful affections of the nerve

not due to any cause outside it. The two varieties may be distinguished as secondary and primary sciatica. Primary sciatica is commonly regarded as a neuralgia. This view is probably in the main erroneous; the vast majority of cases of sciatica are really cases of neuritis of the sciatic nerve. The reasons for this opinion are given in the section on the pathology of the disease.

CAUSES.—Sciatica is far more frequent in males than in females; the proportion has been very variously estimated. It is difficult to obtain statistics on the malady that are free from sources of fallacy, since comparatively few cases enter general hospitals. The incidence of severe sciatica seems to be fairly presented by the 137 cases that were treated in the Devonshire Hospital, Buxton, during the year 1883, and there is no reason to suppose that slight cases differ, in this respect, from those that are severe. The percentage of males is 82; of females, 18; giving a ratio of 4 to 1, which is probably very near the truth. The disease is unknown in childhood, and rare in the second decade of life. It is most frequent between forty and fifty, next between fifty and sixty, and next between thirty and forty. The percentage distribution of the 137 cases at Buxton is as follows:—Ten to twenty, 3 per cent.; twenty to thirty, 9 per cent.; thirty to forty, 19 per cent.; forty to fifty, 29 per cent.; fifty to sixty, 26 per cent.; sixty to seventy, 13 per cent.; over seventy, 1 per cent. The influence of age is nearly the same in each sex.

Two constitutional conditions are potent factors in the production of sciatica,—gout, and that form of rheumatism which affects chiefly the fibrous tissues of the muscles. These two constitutional states cannot always be entirely separated, but many of the sufferers are of opposite types; some are stout, plethoric, distinctly gouty; others pale, weakly, and rheumatic. It is, I think, most common among those who are gouty but do not suffer from articular gout. Such individuals often have many attacks. Gouty inheritance is sometimes to be traced in those who suffer early from the disease. Syphilis has been supposed in some cases to be the cause of a sciatic neuritis, but cases in which the syphilitic nature of the disease is certain are extremely rare.

An exciting cause is to be traced only in a minority of the cases. Exposure to cold is the most common. It is usually local exposure, as by wet boots, standing in water, &c.; sometimes, however, a general chill of the body determines an attack. The exposure to cold may be even more direct, as by sitting on wet grass. I believe that draughty water-closet seats are answerable for some cases. The neuritis occasionally arises by the extension to the nerve of adjacent rheumatism. Thus I have known an attack of that form of lumbago which is localised in the fibrous attachments of the muscles at the back of the sacrum, to pass into sciatica, distinctly by the rheumatic affection of the fibrous tissues, extending along the fasciæ to the nerve-sheath in the neighbourhood of the sciatic notch. Mechanical causes also sometimes excite the disease, and still more often co-operate with other influences.

The pressure of the edge of the chair, in those who sit much, is the most frequent mechanical cause. Muscular over-exertion has been thought occasionally to excite the disease. If the nerve is already tender, a strong contraction of the muscles at the back of the thigh, especially when the knee is flexed, and the muscles can freely shorten and widen, may produce acute pain in the nerve, evidently by its compression. It is conceivable, therefore, that this cause may be effective in a predisposed person. Various morbid processes within the pelvis may cause symptoms of sciatica, usually by acting on the sacral plexus. Thus rectal and other tumours, pelvic inflammation, and injury during labour are occasional causes. A loaded rectum has been thought to be capable of producing it. Mechanical congestion of the plexus of veins which lies over the sacral plexus of nerves is usually regarded as an occasional cause, but this is somewhat hypothetical, and its efficiency can scarcely be demonstrated. Lastly, the sciatic nerve may be secondarily involved in mischief that is outside the pelvis. The most frequent cause of this is disease of the bone, and especially disease of the hip-joint. In all these forms of secondary sciatica the symptoms are due partly to pressure on the nerve, partly to inflammation excited in it by the pressure, or extending to it from the adjacent disease.

PATHOLOGICAL ANATOMY.—Sciatica being a disease that does not itself cause death, observations of the morbid changes are few. In most cases that have been examined, distinct evidence of neuritis has been found, chiefly involving the nerve-sheath, but extending in some cases to the interstitial tissue. The changes are those that have been already described in the account of neuritis (p. 54). In recent cases there is swelling and redness of the sheath, sometimes there are small hæmorrhages, and, in severe cases, similar but slighter alterations in the interstitial tissue, with secondary damage to the nerve-fibres. The signs of inflammation are most intense at the sciatic notch, and opposite the middle of the thigh. They may be limited to one or both of these places, or they may be greatest there, and extend, in slighter degree, over a considerable tract of the nerve.

SYMPTOMS.—The chief symptom of sciatica is pain along the course of the nerve-trunk, often also along that of its branches, and sometimes in the area of its distribution. The affection may begin suddenly, especially in cases of rheumatic origin,—as suddenly as lumbago. More frequently the onset is gradual; slight pain is felt along the back of the thigh, on movements and in postures that make the nerve tense, or cause pressure upon it. As the disease increases, the pain is more readily produced, and becomes greater in degree, until at last the patient is free from induced pain only when at rest and the leg is in a certain position. Any movement that makes the nerve tense causes extreme suffering, and to avoid this, the knee, in walking, is kept slightly flexed, and the leg held stiffly so as to avoid painful changes in the tension of the nerve. As the pain on movement increases, spontaneous pain is added, at first chiefly felt in the nerve-trunk, but soon spreading

to its branches and distribution. It is usually most intense in certain spots—(1) above the hip-joint, near the posterior iliac spine, (2) at the sciatic notch, (3) about the middle of the thigh, (4) behind the knee, (5) below the head of the fibula, (6) behind the external malleolus, (7) on the back of the foot. The pain may radiate over the whole distribution of the nerve, but it is often so distinctly limited to the course of the trunk and branches, that the patient points these out with exactness, when he indicates the course of the pain. It may be dull or acute, and is often burning in character. It may seem to dart downwards, often starting from the highest point behind the iliac bone. As the pain on movement increases, the nerve-trunk becomes extremely tender to pressure, especially along the back of the thigh. Even before the tenderness becomes considerable it may often be elicited in the following manner. Let the patient sit on a chair with the knee at a little more than a right angle. If the finger is then pressed into the popliteal space, so as to make the nerve a little more tense, a pain is felt, shooting up the course of the nerve at the back of the thigh. This is a very useful test of sensitiveness of the nerve, and is of diagnostic value in slight cases. It is not common for tender points to be developed in the periphery.

Abnormal sensations other than pain are often felt in the area of distribution of the nerve, tingling, formication, and the like, and in severe cases there may be irregular spots of anæsthesia on the back of the thigh, on the leg, or the foot. The affection of sensibility at the back of the thigh indicates that the disease extends up the nerve, above the sciatic notch, to the origin of the small sciatic, or that this is involved in a simultaneous neuritis. In severe cases the muscles supplied by the nerve become flabby, tender to the touch, and sometimes distinctly weak and wasted. This is chiefly noticeable in the calf muscles. There may be slight alteration in the electrical irritability, but this amounts to a distinct degenerative reaction only in very severe cases. Slight fever and corresponding constitutional symptoms may attend an onset that is acute, or an inflammation that is intense. Chronic cases, however, are usually not attended by elevation of temperature.

The duration and severity of the affection are extremely variable. It may be trifling in degree, causing pain on movement only, and this may pass away in the course of a few weeks. On the other hand, the spontaneous pain may be so continuous and intense that sleep can be obtained only by the help of narcotics, and the disease may last for many months, and even for a year. Improvement is shown by the subsidence of spontaneous pain, followed by the slow diminution of the pain on movement, and of the tenderness of the nerve. The muscular wasting, that occurs in severe cases, may last long after the active stage of the disease in the nerve, and fibrillary contractions in the muscles that have been affected may continue for years, and are sometimes accompanied by a tendency to painful cramp. The disease

is prone to relapse and still more prone to recur after recovery. A second attack may occur in the same or in the other leg, but both legs are scarcely ever affected at the same time.

The cases of secondary sciatica depending on disease outside the nerve, compressing or irritating it, differ, in some respects, from the primary form. Pain is felt less in the nerve-trunk than in its distribution. The difference is not absolute; it is one of degree. There is rarely, however, the tenderness of the nerve that is so common in primary sciatica. The course of the secondary cases is also different, and depends on the course of the original disease.

Complications of sciatica are rare, but one or two deserve mention. Cutaneous eruptions, usually herpetic in character, have been occasionally met with. These sometimes heal slowly, but, in themselves, they are unimportant. A graver complication, but happily a rare one, is a tendency of the mischief to ascend the nerve. Thus symptoms may spread from the region of the sciatic to that of the lumbar plexus; the pain may spread to the front of the thigh, and the extensors of the knee may become flabby and weak. This is probably due to the passage of an ascending neuritis up the lumbo-sacral cord. In extremely rare cases, the morbid process has apparently reached the spinal cord, and indications of cord disease have thus succeeded those of a primary and apparently simple sciatica.

PATHOLOGY.—The nature of the disease has been already indicated. The evidence that the disease is a neuritis, and not a neuralgia, is chiefly threefold. The conditions under which the disease occurs are very different from those that attend unquestionable neuralgia in other situations. This has been remarked with surprise, even by those who regard it as a neuralgia, but who have failed to see the significance of the difference. Secondly, the neuritic nature of the affection is clearly indicated by the facts of pathological anatomy, scanty though these are. Thirdly, the symptoms in severe cases are proof of the existence of neuritis, the wasting of the muscles and the anæsthesia indicate structural damage to the nerve-fibres, and preclude any other explanation. There is every gradation between these severe forms and those which are slight, and the symptoms in the latter are identical with those of the early stage of the severe cases.

The symptoms find their explanation in the character of the morbid process. The pain in the nerve and its tenderness must be due to irritation of the *nervi nervorum*. The pain referred to the distal portions of the nerve is due to the irritation of the proper fibres by the interstitial inflammation, while their greater damage explains the anæsthesia and muscular wasting.

It may seem strange that, when there is irritation of the nerve-roots or sacral plexus by pelvic disease, or of the trunk by hip-disease, any pain should be felt along the course of the nerve. It might be thought (and has indeed been asserted) that all the pain should be referred to the peripheral distribution of the nerve. The explanation is that the

“*nervi nervorum*” are derived from their own trunk. The nerve itself is part of the area of distribution of its own fibres.

DIAGNOSIS.—The diagnosis of sciatica rests on the position of the pain—on the relation of this to the trunk of the nerve, and to its area of distribution. But the recognition of this relation does not alone suffice for the diagnosis. We have to consider the distinction from other pains in the sciatic area, to discriminate, if we can, sciatic neuritis from sciatic neuralgia, and to decide whether the affection of the nerve is primary, or is secondary to mischief outside it.

Branches of the sciatic nerve and sacral plexus ramify over the hip-bone, and pain in sciatica may be felt near the hip-joint; hence disease of this joint and sciatica may be confounded. The pain in simple disease of the hip-joint does not extend down the back of the thigh in the course of the sciatic, and there is no tenderness of the nerve. The danger of this error is not great, if a careful examination is made, and has been much exaggerated by writers unfamiliar with the characteristic symptoms of true sciatica.

The distinction between a sciatic neuritis and a sciatic neuralgia is sometimes difficult, although less frequently than might be inferred from current accounts of these diseases, in which the history of the neuralgia has been written from the symptoms of the neuritis. If we recognise that all cases of sciatica with persistent tenderness of the nerve, are really neuritic, cases of sciatic neuralgia become extremely rare. The two diseases occur usually under different conditions: the subjects of neuralgia have often suffered from neuralgia elsewhere, and are generally weakly and anæmic. The pain is from the first spontaneous; posture has little influence upon it; movement is not itself painful, although it may excite paroxysms of pain. The pain is referred to the branches and distribution of the nerve rather than to its trunk, and tenderness of the nerve, if it exists, is altogether subordinate to the spontaneous pain.

Secondary sciatica is usually produced by disease of bone about the hip-joint, or of the joint itself, or by disease in the pelvis. In the former case, a careful examination (never to be omitted in any case of sciatica) at once reveals the mischief. When the disease is within the pelvis, the tenderness of the trunk of the nerve is slight in proportion to the pain, and this circumstance should always lead to a careful search for any indication of pelvic mischief. In any case of doubt, a rectal examination should be made.

Sciatic pain occurs in some diseases of the bones of the spine, in lesions of the cauda equina, and occasionally in disease of the spinal cord itself. In these cases we have no tenderness of the nerve; the pain is chiefly peripheral, and very often bilateral. Double true sciatica is almost unknown, and bilateral pain should always suggest disease of the nerve-roots (see p. 83). The pains of locomotor ataxy are often felt in the sciatic area, and occasionally follow the course of the sciatic nerve, but their wider range, their fugitive character, and

their association with other symptoms of tabes, should prevent an error.

PROGNOSIS.—The prognosis of sciatica, not dependent on disease outside the nerve, is always good as regards ultimate recovery. As a general rule, the probable duration of the disease is proportioned to the severity of the symptoms. The practicability of adequate rest is an important element in the prognosis. Irritating exertion may lengthen the duration of the disease by many months.

TREATMENT.—The principles of the treatment of sciatica are those of neuritis already described, and only the points of special importance need be here repeated. In all cases rest to the limb is of the utmost importance, and its urgency is proportioned to the severity of the symptoms. A slight attack may pass away, in spite of a moderate amount of standing and walking, but many slight cases are converted into severe ones by unwise exertion. All postures and all movements which increase pain should be avoided as far as possible. The same principle applies to mechanical compression of the nerve by hard seats, and by strong contractions of the flexors of the knee. In more positive treatment, the possible causes of the disease must be remembered. In gouty cases saline purgatives are often of signal service, and are distinctly useful in preventing attacks in those who are liable. In the acute stage of a severe attack, hot linseed-meal poultices should be applied along the course of the nerve, and small doses of mercury may be given with advantage. Counter-irritation is of great value, and cannot be employed too early. A commencing attack may often be cut short in a few days by rest, and a series of mustard plasters or small blisters applied over the seat of pain, as this changes under their influence, chasing it, as it were, from one spot to another, until it disappears. Spontaneous pain can only be relieved by sedatives. Morphia is the surest, but often an injection of half a grain of cocain relieves the pain as effectually, and is far less objectionable. Cocain must be injected at the seat of the pain. The injection of morphia may be made, with advantage, over the inflamed part of the nerve, so as to combine cutaneous acupuncture with the sedative influence. Simple acupuncture along the course of the nerve has been recommended; it gives temporary relief to the sciatic pain, as does any superficial pain, however produced, but the cases are very few in which it has a permanent effect. Sedative liniments and ointments may also be applied along the course of the nerve; the most useful are belladonna liniment mixed with an equal part of chloroform liniment, and aconite ointment, rubbed in until distinct tingling is produced. Electricity is chiefly useful in the later stages; its method of use has been described in the account of the treatment of neuritis. In very obstinate cases, nerve-stretching has done good; sometimes, perhaps, by releasing the nerve from adhesions, but probably more often by effecting an energetic counter-irritation, and enforcing a beneficial rest.

MULTIPLE NEURITIS.

The term "multiple neuritis," or "polyneuritis," is applied to the condition in which many nerves are inflamed simultaneously or in rapid succession. It has also been called "disseminated neuritis," but this is an inappropriate name because a single nerve may be the seat of disseminated inflammation. The discovery that certain combinations of symptoms, formerly thought to depend on disease of the spinal cord, are really due to disease of the peripheral nerves, is one of the most important steps in the recent advance of neurology. It has profoundly modified many of our conceptions, not only of the processes of disease, but of the range of action of certain morbid influences.

The occurrence of multiple neuritis was first demonstrated by Duménil, of Rouen (1864), although the leprous form had been previously described by Virchow. Graves, indeed, long ago suspected that many cases of paralysis were due to disease of the nerves, but he based his opinion on the normal aspect of the spinal cord; and in most of the cases he describes it is probable that modern methods of examination would have revealed central disease. Duménil's observations attracted little notice, and it was not until fresh facts were brought forward by Joffroy (1879), Leyden (1880), and Grainger Stewart (1881), that attention was generally directed to the subject. During the last five years a large number of observations have been published, especially by Eichorst, Eisenlohr, Pitres, and Schulz.*

Multiple neuritis presents the same varieties that have been described in the general account of inflammation of the nerves. The morbid change may be interstitial, or parenchymatous and degenerative, or both. The pathological forms that occur cannot yet be adequately defined, but five chief classes may be distinguished. (1) Diphtheritic neuritis; acute parenchymatous degeneration of the nerves occurs after diphtheria. The changes are not, however, confined to the nerves, and the condition is considered in a later part of this volume. (2) Tabetic neuritis; in locomotor ataxy degenerative changes are common in the peripheral nerves; they are usually very chronic, and it is perhaps doubtful whether it is correct to regard them as inflammatory, although they are identical in character with forms which, when acute, are commonly so regarded. Except in their relation to some other forms of neuritis, the tabetic changes will not be considered in this section. (3) Leprous neuritis; the slow interstitial overgrowth of connective tissue which occurs in anæsthetic leprosy. (4) Endemic neuritis, such as occurs in the curious and terrible endemic disease of Japan called kak-ké. This form appears to be due to endemic influences, and it is probable

* A useful summary of our knowledge, with many original cases, will be found in Dr. Buzzard's Harveian Lectures, published in the 'Lancet' for November and December, 1885.

that some other cases of neuritis may be due to a similar cause. (5) We must at present place together the other forms of multiple neuritis common in this country. They differ somewhat in pathological characters, in the acuteness of their course, in the symptoms they produce, and the causes to which they are due. Nevertheless no constant varieties have yet been ascertained. This group will probably ultimately be subdivided, at present we can only distinguish the tendencies of its varieties and the difference of its causes. In the following account the statements made apply to this group generally; endemic and leprous neuritis will be separately described at the end of the section.

ETIOLOGY.—Primary multiple neuritis affects both sexes, but, in this country at least, females are more liable to suffer than males. The disease is one of adult life, and is scarcely ever met with until the age of twenty-five is passed. It is most common between thirty and fifty, but has been met with as late as sixty. Its most frequent cause, beyond all question, is chronic alcoholism. This lesion is the common cause of alcoholic paralysis. For some reason, at present unknown, females suffer from this cause far more frequently than males. I have notes, for instance, of eight cases of characteristic alcoholic neuritis in females, and not of one in a male. The fact is remarkable considering that male drinkers are certainly more numerous than female drinkers. Apart from alcoholism, the disease is probably more common in males, but the influence of this cause is so great as to determine a preponderance of females when all cases are taken together. In cases in which this cause is not operative others can sometimes be traced: exposure to cold, extreme and long-continued fatigue, exhaustion after other maladies, acute and chronic. In a few instances the symptoms have followed acute or subacute articular rheumatism, but the disease has sometimes been ascribed to rheumatism on insufficient grounds, since rheumatoid pains, referred in part to the joints, are often an early symptom of neuritis. Nevertheless, true rheumatism has been an apparent cause in some cases, and, as we have already seen, single nerves may be affected from the same influence. Many sufferers from multiple neuritis have died from phthisis, but the nature of the association of the two diseases is obscure.

It is probable that some blood-states allied to septicæmia may cause multiple neuritis. Thus, a man, thirteen days after a stab-wound under the left clavicle, which healed well, had an attack of parotitis with facial palsy; on the fortieth day there developed paralysis of the tongue, vocal cords, and limbs, and on the sixth day after the onset of these symptoms he died from respiratory palsy. Extensive peripheral neuritis was the only nerve-lesion.* In some instances more than one cause is operative; exhaustion and exposure to cold may concur with alcoholism.

PATHOLOGICAL ANATOMY.—The changes in the nerves are those

* Roth, 'Correspondenzbl. f. Schweizer Aerzte,' 1883, No. 13.

already described in the account of neuritis. They vary, however, very much in different cases. In some, the inflammation chiefly affects the sheath and interstitial tissue; in other cases both the connective tissue and nerve-fibres are involved, as in Fig. 39, p. 54. This was the condition in the case of presumably septicæmic neuritis mentioned above. In others again, the fibres are affected chiefly or exclusively; they present the degeneration which when acute is regarded as indicative of parenchymatous inflammation. We cannot, at present, connect these pathological varieties with any differences in causation. The more acute the case, the more probable is it that the connective tissue as well as the nerve-fibres will be involved.

Naked-eye changes are present only when the connective tissue and sheath are inflamed in considerable degree. In recent and acute cases of this character, the nerves may be found reddened, swollen, and sometimes small hæmorrhages are visible. In older cases they may or may not be swollen, but are usually softened, and even pulpy. When the changes are confined to the nerve-fibres, diminished consistence may be the only alteration that can be recognised with the unaided eye. In the former cases, the microscope shows the sheath to be infiltrated with lymphoid cells (probably leucocytes), and in old cases many spindle-cells and fibres increase its bulk. The same changes may be traced in the septa between the fasciculi, and in the secondary sheaths that surround the latter. The walls of the vessels are also thickened (Fig. 52, A). In recent cases, the small vessels may be surrounded by leucocytes. The fibres themselves are always damaged in some degree, and as in the case from which Figs. 51 and 52 are drawn, they may suffer chiefly, the connective tissue presenting comparatively little change. In this case, the patient was a woman, aged 33, alcoholic; the course of the disease was subacute; the symptoms reached a considerable degree in a few weeks, and the patient died, chiefly from coincident liver disease, five months after the onset. The symptoms were characteristic, and continued up to the time of death. The condition found after death is a good illustration of the parenchymatous or degenerative form, but in all forms of neuritis the process in the nerve-fibres is the same, whether the connective tissue is or is not much affected. The fibres undergo acute degeneration, essentially the same as that which occurs after an injury to a nerve. It has been represented in Figs. 28 (p. 40), and 29 (p. 42), and, associated with inflammation of the sheath, in Fig. 39. Examination in the recent state reveals abundant products of the degeneration of the fibres, granule corpuscles, &c. After hardening, a transverse section, stained with carmine, and cleared in the usual way, presents the appearance shown in Fig. 51, A. There is a slight increase in the connective tissue between the fasciculi; the area of these is occupied by tracts of connective tissue, which under a higher magnifying power (B) have a branching form, enclosing spaces which have been occupied by nerve-fibres, and in some of which healthy fibres still

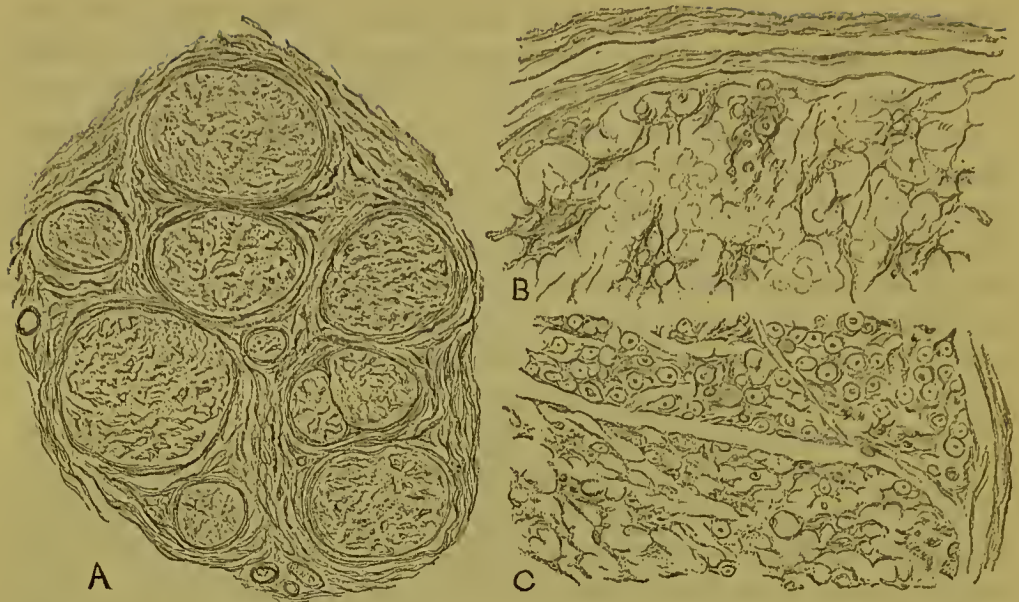


FIG. 51.—Multiple alcoholic neuritis: sections stained with carmine and cleared. A. Transverse section of part of sciatic nerve; low power. B. Part of a fasciculus of same more highly magnified. C. Part of a less affected fasciculus from a musculo-spiral nerve.

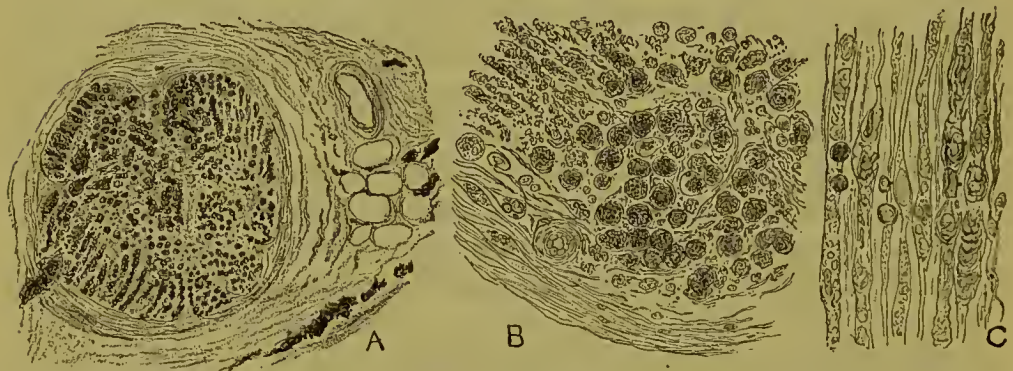


FIG. 52.—Sections from the same sciatic nerve, stained with osmic acid and mounted in glycerine. A. Fasciculus under low power. B. Part of a fasciculus more highly magnified. C. Longitudinal section of nerve-fibres. (From sections prepared by Dr. Beevor.)

remain (c). The space between this tissue is occupied by the products of degeneration of the fibres, but these are rendered invisible by the clearing process, and only faint indications of their outline can be seen (B). In sections stained with osmic acid, and mounted in glycerine (Fig. 52) these products of degeneration, stained dark, are conspicuous. Under a low magnifying power (A), a fasciculus is seen to be studded with black granular spots, elongated where the fibres are divided obliquely; the increase in the tissue between these degenerated fibres is distinct; aggregations of myelin are seen in the connective tissue between the fasciculi. Under higher powers (B), the dark spots are seen to be the sections of degenerated nerve-fibres, in some places two or more being blended into a larger mass. In a longitudinal section (c), the process of degeneration of the fibres is

more distinctly seen, and from it the appearance of the transverse section can be better understood. The white substance is broken up and enlarges the fibre at some places, while at others it has been removed, and the sheath is left empty. Round masses of myelin lie (on the left) outside the fibres. The nuclei of the fibres are enlarged; one, of considerable size, is seen in the centre of the figure. Connective-tissue fibres and cells lie between the nerve-elements, and near the sheath are round and spindle cells containing myelin granules and globules which they have taken up (B).

The distribution of the changes in the nerves varies much in different cases. The signs of inflammation of the sheath may exist only on the larger and medium-sized nerves of the limbs, and in the smaller nerves the changes may be confined to the nerve-fibres. These may be traced down into the intramuscular nerves and their endings. In some of these cases, the latter may be secondarily degenerated. On the larger nerves, the signs of inflammation of the sheath are most intense at certain spots, especially (as in simple neuritis) where a nerve turns round a bone or passes through a fascia, or divides. When the trunks are traced upwards, the evidence of inflammation of the sheath, as well as the change in the fibres, becomes slighter, and in all forms, the anterior roots are usually normal.

In cases in which the affection is chiefly degenerative, the changes are always intense in the peripheral parts of the nerves, and usually lessen when the larger trunks are reached, becoming progressively slighter, and the proportion of normal fibres larger, until the morbid appearance ceases. The centripetal extent of the disease, the degree in which the larger trunks are affected, varies according to the duration and severity of the case. In the case from which the illustrations are taken, even the sciatic nerve presented few healthy fibres. But as usual, the anterior roots were healthy.

The nerves of the limbs alone suffer in the majority of cases. The corresponding nerves on the two sides are usually affected. The musculo-spiral nerve in the arm and the anterior tibial nerve in the leg usually suffer first and in greatest degree, but often all the nerves of the limbs are involved. The nerves of the trunk are affected only in severe cases. Very rarely the phrenic, pneumogastric, facial, and hypoglossal have been inflamed. The other cranial nerves escape.

The *muscles* present changes of the same character as those which result from simple degeneration (see p. 43). They are paler than normal and smaller in bulk. The fibres are reduced in size, and pale; the transverse striation may be preserved, or they may be granular; normal fibres may be found side by side with those that are degenerated. The nuclei of their sheaths may be increased in number, and granular, and pigment masses may accumulate between the fibres.

The *spinal cord* has been found healthy in most cases. The ganglion-cells, in some instances, have presented abnormal appearances, a

vitreous appearance or vacuolation, and this when the anterior nerve-roots were not diseased. Now and then more considerable changes, usually those of chronic myelitis, have been found in the cord. They are probably merely coincident.

Morbid appearances in other organs are common in the cases of alcoholic origin. The brain may present signs of chronic meningitis. The liver is frequently, and the kidneys are occasionally, found diseased.

SYMPTOMS.—The symptoms of multiple neuritis resemble in their characters those produced by inflammation of single nerves, differing, however, in their wide range and special distribution. The manifestations of the disease are, in most cases, very characteristic, in spite of the fact that the extent of the symptoms obscures that relation to individual nerve areas which constitutes so important a feature of the inflammation of single nerves. The early and dominant symptoms are those of motor palsy and sensory irritation. The distribution of the palsy is especially determined by the tendency of the musculo-spiral and anterior tibial nerves to suffer most. In consequence of this there is a symmetrical paralysis of the extensors of the hand and flexors of the foot, with resulting wrist-drop and foot-drop (Fig. 53). The sensory irritation causes pain and tenderness, usually localised in the nerve-trunks. The progress of the disease is attended by more profound indications of nerve damage, wasting of muscles and impairment of sensation in the distribution of the most affected nerve-trunks.

The onset of the symptoms may be acute or subacute, but is rarely so slow as to deserve the name of chronic. An acute onset may be attended by considerable constitutional disturbance, rigors, fever, &c., and this may last for from one to three or four weeks. The initial pyrexia may amount to 103° or 104° . The first definite symptom is usually a sensation of tingling, or "pins and needles," in the extremities, chiefly in the fingers and toes. It may or may not be preceded by vague rheumatoid pains, but to it pain is soon added. This is commonly at first dull in character, but speedily increases to a considerable degree, and becomes more acute—sharp, darting, or burning in character. It may be felt along the course of the nerve-trunks or in the area of their distribution, and is sometimes especially severe in the fingers and toes. It is often increased by movement. Occasionally, in very chronic cases, the amount of pain is trifling. With the pain there develops considerable tenderness of the limbs, increased sensitiveness of the skin and deeper structures. The nerve-trunks usually become extremely tender, and sometimes the more accessible nerves, such as the ulnar, may be felt to be distinctly swollen. The muscles become very sensitive to pressure, whether they are the seat of spontaneous pain or not. This tenderness of the muscles is even more constant than that of the nerve-trunks. Muscular weakness is soon added to the sym-

ptoms of sensory irritation, although for a time it may be masked by the interference with movement caused by the pain. The loss of power usually begins in the arms as deficient power of extension of the wrist and fingers. Both arms are affected together or in quick succession. The palsy resembles that which results from lead-poison-



FIG. 53.—Multiple alcoholic neuritis; palsy of extensors of wrist and flexors of ankle. (From a photograph by Mr. Hyde Marriott, B.Sc.)

ing, but the supinator longus is usually involved. With, or soon after, extension of the hand, the power of flexion of the ankle and extension of the toes becomes impaired. Sometimes the legs suffer before the arms, and occasionally the symptoms are confined to the lower limbs. The flexors of the hand and extensors of the foot suffer less than their opponents, but in cases of greater severity they also become weak. In these cases also the muscles moving the elbow and knee, and even those of the shoulder and hip, become feeble, but lose all power only in very severe cases, and in these the trunk muscles may be involved. The diaphragm may be paralysed; constant frequency of pulse and paralysis of the vocal cords may indicate a neuritis of the vagus (Roth, Vierordt); and the tongue and face may become affected (Strube). The affected muscles soon become flabby and waste. The nerves lose their electrical excitability, and the muscles present, in most cases, the reaction of degeneration. The increase of voltaic irritability may be slight in the cases of chronic course, and even in acute cases the early increase gives place to depression much sooner than in cases of focal neuritis or of infantile paralysis. It is often very difficult to make a satisfactory electrical examination, on account of the extreme sensitiveness of the limbs. Besides motor weakness, there is sometimes distinct inco-ordination of movement, which may closely resemble that of locomotor ataxy.

The hyperæsthesia of the skin and deeper structures usually continues for a long time, often through the entire course of the disease. It is extensive in most cases, but has been known to be limited to the extremities, and even to the palms and soles. As the nerve-fibres suffer in greater degree, the sensibility of the skin is usually impaired, that to touch first and chiefly. Increased sensitiveness to pain often

coexists with anæsthesia. The loss of sensation is always greatest on the extremities, but may extend up the limbs, first along the outer side of the leg, and the radial side of the forearm. It is rare for sensibility to pain to be lost, but its conduction has been delayed in some cases.

Reflex action in the affected limbs is always lost. The myotatic irritability disappears early. The knee-jerk may be lost even before the weakness of the extensors of the knee has reached an appreciable degree. Trophic changes in the skin, nails, and hairs are common, similar to those met with in ordinary neuritis. Œdema of the limbs is frequent; probably, in alcoholic cases, the blood-state conduces to its occurrence. In one case the œdema existed only over the nerve-trunks, in the neighbourhood of joints (Leyden). Effusion into joints has been observed, but there is some doubt whether this was the consequence of the neuritis, or was rheumatic and primary. Slower changes in the nutrition of the joints are common, and may lead to adhesions. The sphincters are affected only when there is also disease of the spinal cord.

In rare cases the motor weakness is very slight, the sensory symptoms exist chiefly in the periphery, there is little tenderness of nerve-trunks, and the inco-ordination, already described as occasionally present, is a conspicuous symptom. Such cases present a close resemblance to the symptoms of locomotor ataxy, and the condition has been termed "alcoholic pseudo-tabes,"* or "alcoholic ataxia."†

The *course* of the affection varies according to its acuteness and severity. In cases of moderate degree, the symptoms increase during four or six weeks, then become stationary, and, after one or two months, slowly improve. The pains lessen, but the hyperæsthesia may continue for some time after the pains have ceased. Power slowly returns, first in the muscles affected last and least, and afterwards in those paralysed and wasted in greater degree. In these the weakness lasts for many months, and their opponents are apt to become shortened. The contracture of the calf muscles, secondary to palsy of the flexors of the ankle, constitutes a grave hindrance to the use of the legs. The palsy of the arms usually lasts longer than that of the legs. Often, for instance, at the end of six or eight months, the legs have recovered and the extensors of the wrist and fingers are still feeble. Sometimes power returns first in the arms. Improvement goes on for a long time, but occasionally there is some permanent loss of power. It is remarkable, however, how much improvement may take place, even after the paralysis has been considerable for a year. Relapses are apt to occur, but only when the cause of the neuritis is still in operation.

On the other hand, the disease may continue to increase for several months, or may develop with such acuteness and intensity that at the end of ten days or a fortnight there is complete paralysis of the limbs ;

* Krucke, 'Deut. med. Zeitung,' 1884, No. 72.

† Dreschfeld, 'Brain,' July, 1884, p. 201.

the intercostals share the weakness of the muscles of the trunk, and, if the diaphragm also becomes paralysed, death may occur from the failure of breathing power. Such cases have been known to result in death at the end of six and ten days. In other, slightly less intense, cases, the patient has lost all power of movement, and has died at the end of a few weeks or months, worn out by continued pain and bedsores, or suffocated by a trifling bronchitis in consequence of the partial palsy of the respiratory muscles. In many cases, however, the immediate cause of death has been associated disease, either phthisis, or some other consequence of alcoholism, especially disease of the liver. I have twice known rapid and unexpected cardiac failure to cause death in alcoholic cases. It may have been paralytic in nature, but as each patient had recovered from the graver symptoms, it is more likely to be due to degeneration of the muscular tissue of the heart.

Of *complications*, the most important, beyond those that have just been named, is mental derangement. This is confined to the alcoholic cases. It may be acute or subacute, and consists in failure of mental power, often accompanied by delusions. It is ultimately recovered from, if the patient's habits can be reformed. When there is no pronounced mental derangement there is often in women a marked incapacity for endurance, a tendency to exaggerate suffering, and an intolerance of remedial measures, which increases very much the difficulty of dealing with these cases.

PATHOLOGY.—The changes found in the nerves afford a complete explanation of all the symptoms of the disease. The ataxy observed in some cases may be due to damage to the sensory muscle-nerves. The relative affection of the nerve-sheath and nerve-fibres cannot at present be explained. The relation to alcohol suggests that the neuritis is the result of a direct toxic influence on the nerve-elements themselves. It has been indeed assumed that the influence is exerted on special trophic centres in the spinal cord which govern the nutrition of the nerve-fibres (Erb), but the existence of such centres is not only not proved but is improbable. The inconstancy of the slight changes in the ganglion cells of the spinal cord, and the normal state of the anterior roots, preclude the assumption that the intense degeneration of the nerve-fibres can be the result of those alterations, and make it probable that these central changes, when present, are an associated effect of the same cause. The severe general symptoms that occasionally attend the onset has suggested the idea that the malady is often of the nature of an acute specific disease. The gradation that exists between these and more chronic forms is an objection to this theory ; nevertheless the hypothesis must not be thrown altogether aside, since multiple neuritis in the Japanese kak-ké and in leprosy seems to form part of a specific endemic malady, and some other blood-states may produce it. On other grounds the occurrence of multiple degeneration in the peripheral nerves from the influence of alcohol is a fact of great interest in con-

nection with the pathology of locomotor ataxy. We have seen that the symptoms may closely resemble those of tabes. In this disease the morbid influence acts on the sensory nerves only. It seems probable that some influences may act chiefly on the motor or on the sensory nerves, the incidence being perhaps determined in part by predispositions, at present obscure.

DIAGNOSIS.—The diagnosis of multiple neuritis depends on the combination of motor and sensory symptoms above described, on their localisation in the distal parts of the limbs, and on the tenderness of the nerve-trunks and of the muscles. The early pains are often mistaken for those of rheumatism, but their seat is not that of rheumatic pain, and their association with tingling in the extremities should excite a suspicion that they are of nerve origin, even before muscular weakness renders their nature clear. The symptoms resemble most closely those of certain diseases of the spinal cord, acute and subacute inflammation of the grey matter, polio-myelitis. Until recently, indeed, all cases of multiple neuritis were looked upon as spinal. In each disease there are a febrile onset, muscular wasting with the reaction of degeneration, initial rheumatic pains, and a tendency to the spontaneous recovery of the least affected parts. The distinction rests on the symmetrical localisation of the neuritic palsy, while that of polio-myelitis is characteristically random in distribution; on the persistence and severity of the neuritic pains; on the tenderness of the inflamed nerve-trunks; and on the anæsthesia in the area of their distribution, a symptom never present in polio-myelitis. Pachymeningitis, damaging the nerve-roots, may cause both palsy, wasting, and anæsthesia, but in this all four limbs are not affected, and the legs rarely suffer; the anæsthesia extends on to the upper part of the limbs and trunk; there is no tenderness of nerve-trunks, and there is usually distinct evidence of damage to the spinal cord itself. A mysterious disease, termed “acute ascending paralysis” may resemble the most rapid form of multiple neuritis, but, in it, the symptoms ascend the trunk to the arms, and do not begin in the hands and feet at the same time, as does the usual form of multiple neuritis. Moreover, there is no anæsthesia in the spinal malady. Diphtheritic paralysis, when severe in degree, may also be attended by muscular wasting and anæsthesia, but there is no considerable pain, and weakness in the limbs succeeds that in the palate and ciliary muscle. The distinction of the pseudo-tabetic form from true tabes is very difficult unless muscular weakness is distinct. There is no affection of the internal ocular muscles, no loss of reflex action in the iris, no affection of the sphincters, and no optic nerve atrophy, but these symptoms may be absent in true tabes. A history of alcoholism, with these indications, affords ground for a presumptive diagnosis, but it must also be remembered that true tabes may occur in an alcoholic subject.

PROGNOSIS.—In the early stage of an acute case there is danger to life in proportion as the upper parts of the limbs and trunk are paralysed, and the respiratory muscles are thus threatened or involved. When the disease has become stationary, the danger to life is not great, except in the severe cases in which the pains are intense, the patient helpless, and an affection of the sphincters or a girdle pain makes it probable that the mischief has extended to the spinal cord. The prognosis depends, however, as much on the complications as on the disease of the nerves. Organic disease of the liver, kidneys, or lungs, always renders the prognosis grave. The extent to which the habits of the patient can be controlled is an important element. If the danger to life has passed, recovery in the parts least affected may be expected to occur in from three to six months; in those in which the muscular wasting is greatest, not for nine or twelve months, and some permanent weakness may be left. It is remarkable, however, how great and how prolonged a loss of power may ultimately be recovered from.

TREATMENT.—At the onset of an acute case, the treatment should be that suitable for a local inflammation or a morbid blood-state. Absolute rest is of great importance. The excitation of the nerves involved in movement is likely to intensify the morbid process in them. Light nutritious food should be given, and the bowels gently opened. If alcohol has been taken in excess, it must be reduced to a small quantity, or, better still, if practicable, it should be given up altogether. This point in the treatment of alcoholic cases is of extreme importance, and often of extreme difficulty. In the case of every female drinker, deception should be considered not only possible but probable. Servants are corrupted, and alcohol is often taken in secret, and absolutely denied. When a case does not present the improvement that might reasonably be expected, this influence will generally be found to be at work. Hence it is of great importance that these patients should be under the immediate care of trained nurses, and not of their own servants or relatives. The special treatment of the disease is the same as that for inflammation of single nerves already described. Warm poultices or fomentations should be applied to the limbs, and a daily warm bath, for twenty minutes or half an hour, is often of great service. The general rules already mentioned govern the use of counter-irritation and electricity. A very gentle voltaic current may be applied to those muscles which present indications of nerve degeneration, but not until the most acute stage of the disease is over. Rubbing is very useful, and may be employed almost from the first; in the early period it should be very gentle; afterwards attempts should be made, by passive movement, to lessen the tendency to contractures. The extreme sensitiveness of the skin and muscles, and the intolerance of the patients, often render local treatment very difficult. Attention to posture will do much to prevent

the occurrence of contractures, but it is often impossible to induce the patient to lie in any other position than that which favours the occurrence of shortening of the muscles. These contractures hinder progress during convalescence. Tenotomy is, however, seldom needed. It is remarkable how much muscular shortening will gradually give way.

During an acute febrile onset, the treatment by drugs should be that of the constitutional state: citrate of potash, nitric ether, and compound tincture of bark are the most suitable, and salicylate of soda is said to do good in the cases which follow exposure to cold. Afterwards iron is useful, with quinine and small doses of strychnine or nuxvomica. Mercury is not so efficient as it is in cases of simple inflammation of a nerve-sheath, and iodide of potassium has not seemed to me to have any special influence on the disease. In chronic cases, or in the chronic stage of acute cases, arsenic may be given with seeming advantage. Cod-liver oil is beneficial in patients whose general nutrition is impaired. The pains may need opium or morphia, by the mouth or injection, but these should be given as sparingly as possible in alcoholic cases. Cocaine sometimes relieves local pain, and hypodermic injections of a 2 per cent. solution of carbolic acid have been found, by Caspari, to give relief. Any complications that may be present, in the nerve-centres or other organs, must receive appropriate treatment.

ENDEMIC NEURITIS.

Multiple neuritis appears, from recent researches, to play the chief part in the production of the nervous symptoms in the terrible malady known as *Kak-ké* in Japan, and as *Beri-beri* in other parts of the East Indies. The inflammation of the nerves involves both the connective-tissue elements and the nerve-fibres. Rapid degeneration of the muscles occurs. The spinal cord also presents changes, but these are very variable in degree, and are thought by most observers to be either secondary to those in the nerves or merely coincident. The disease varies much in severity, and in the most acute cases, almost every tissue in the body suffers. The chief nervous symptoms are paralysis, muscular wasting, pains, and "numbness" or "burning" in the skin; all these symptoms being chiefly seated in the legs. In the acute cases, the nervous symptoms are subordinate to those of the general blood-state. Some form of malarial poison is probably the agent in the production of the disease. It occurs far more frequently in men than in women, in young adults than in children or the old, and in summer than in winter. It is said to be especially prevalent when the first heat of summer falls on a soil saturated with moisture.

Two cases of paraplegia have come under my notice which were possibly due to malarial neuritis. In each case the disease developed

in a fever district, one in India, the other on the East Coast of Africa. The paralysis in each case involved chiefly the muscles in front of the leg, flexors of foot and extensors of toes; these presented well-marked reaction of degeneration; there were pains, but there was no anæsthesia. In each case there was steady improvement under treatment (tonics, electricity, &c.), although secondary contracture in the calf muscles hindered, for a time, the recovery of walking power. The facts that the paralysis was confined to the legs, and that there was no anæsthesia, seem opposed to the opinion that the lesion was neuritis, but these characters obtain also in beri-beri, and it is possible that the neuritis which results from malarial influences may affect chiefly the motor nerve-fibres.

LEPROUS NEURITIS.

Far removed as leprosy may seem to be from the malady last described, both by its general character and extremely chronic course, it is certainly also an endemic disease, and high authorities have sought its cause in malarial and miasmatic influences. The pathology of leprosy has been carried a step further than that of most endemic diseases, since the researches of Carter, Hansen, and Neisser have proved the constant presence of a specific bacillus in all the new formations met with in the disease.

The nervous symptoms of leprosy depend almost exclusively on inflammation of the nerves. They are muscular wasting and anæsthesia, greatest in degree towards the extremities of the limbs. There may be unpleasant tingling sensations, but pains are trifling, perhaps because the process is extremely chronic. Fig. 54 shows the distribution of anæsthesia in the hands in a well-marked example of this disease which came under my observation some years ago. The patient was a creole of Mauritius, who had come to England in early childhood, and presented the first distinct symptoms at twelve years of age—changes in the pigmentation of the skin, anæsthesia, and muscular



FIG. 54.—Distribution of anæsthesia in a case of leprosy. The black areas indicate the loss.

wasting. They steadily increased during the next four years, the anæsthesia extending each half year from one finger to another. There

was also scattered anæsthesia of the legs below the knee. As the figure shows, the loss of sensation does not correspond in area to nerve distribution. This depends on the fact that all the nerves, even those of the fingers, are diseased, but in none are all the nerve-fibres commonly destroyed. The muscular wasting was considerable, although only in the small muscles of the hands was it comparable to that of progressive muscular atrophy. The electrical excitability of the muscles was greatly lowered to each current.

The neuritis of leprosy is typically interstitial (Fig. 55). The

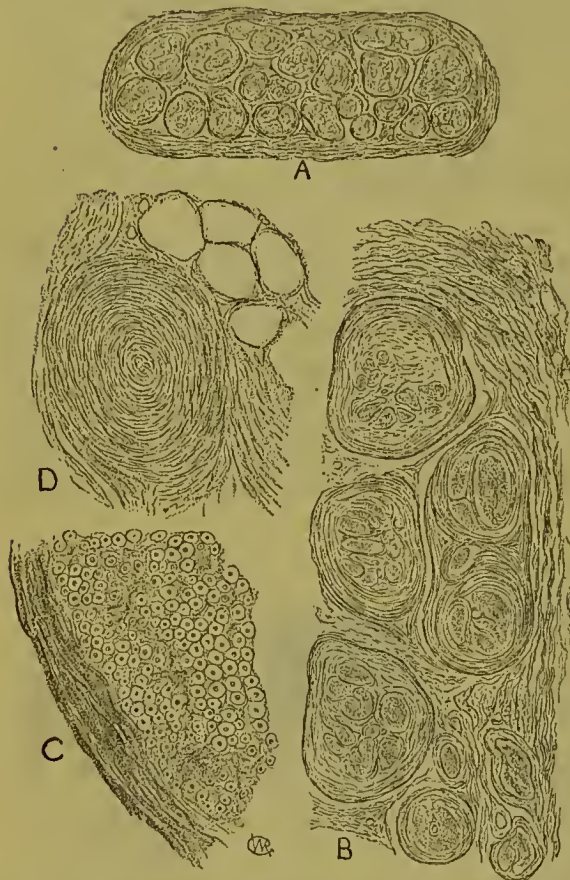


FIG. 55.—Sections of nerves from a case of anæsthetic leprosy, under the care of Dr. Buzzard. A, median nerve at wrist $\times 5$; B, portion of same more highly magnified; C, part of a less diseased fasciculus from the ulnar nerve; D, a small fasciculus from median in which the concentric fibres have invaded the whole area of the fasciculus.

primary sheath and the secondary sheaths of the fasciculi are greatly increased in thickness, and consist of nucleated fibrous tissue arranged concentrically (A, B). From the sheath, tracts extend into the interior of each fasciculus (B), isolating the groups of nerve-fibres. The increase of tissue even extends between the fibres themselves (C) and these undergo slow wasting; many of the fibres in the figure are seen to be distinctly narrower than normal. The concentric growth and fibrous tissue may even invade the whole area of the fasciculus, all the nerve-fibres perishing before it (D). The characteristic bacillus of leprosy is found abundantly, in recent cases, in the new tissue of the nerves. Peculiar cells are met with, infiltrated with the organisms. As the fibrous

tissue develops and contracts, the bacilli seem to perish, and ultimately can no longer be discovered. The fact that the connective tissue of the nerves should be thus clearly one of the structures for which an organised virus has a special affinity, is of extreme interest in connection with the action of the varied causes of multiple neuritis.

PART III.

DISEASES OF THE SPINAL CORD.

INTRODUCTION.

ANATOMY OF THE SPINAL CORD.

THE spinal cord, it will be remembered, is much shorter than the spinal canal, reaching only to the second lumbar vertebra. Hence the nerve-roots descend to their foramina of exit. The lower they arise, the longer is their intra-spinal course. All those below the second lumbar pair leave the canal below the lowest portion of the cord. It is convenient to speak of the portion of the spinal cord from which each pair of nerves arise as the corresponding "segment" of the cord. The segments are longest in the dorsal region, and shortest in the lumbar enlargement.

The only parts of the spinal column that we can feel are the vertebral spines. These are not all on a level with the bodies of their vertebræ. It is important, therefore, to know the relation of the spines to the bodies of the several vertebræ, and of these to the origin of the nerves. These relations are shown in the accompanying figure (Fig. 56) with sufficient clearness to render a detailed description unnecessary.*

Of the *membranes*, the pia mater closely invests the surface, and is continuous with the tracts of connective tissue that pass within the substance of the spinal cord. It is also prolonged along the nerve-roots as their sheaths. The arachnoid forms a much less close investment. The dura mater is not applied to the bones, as it is within the cranium, but a layer of fat and a plexus of large veins intervene between the two. An extension of the dura mater passes along each nerve-root, and blends with its sheath.

STRUCTURE OF THE SPINAL CORD.—The general form of the cord, the enlargements it presents, and its constitution of white and grey substance, are too well known to need description here. The *white*

* The details of the chief relations are enumerated in my 'Diagnosis of Diseases of the Spinal Cord,' 3rd ed., p. 6.

substance surrounds the grey, except at the two points at which the posterior horns come to the surface (Fig. 57). It consists of medullated nerve-fibres, running longitudinally.

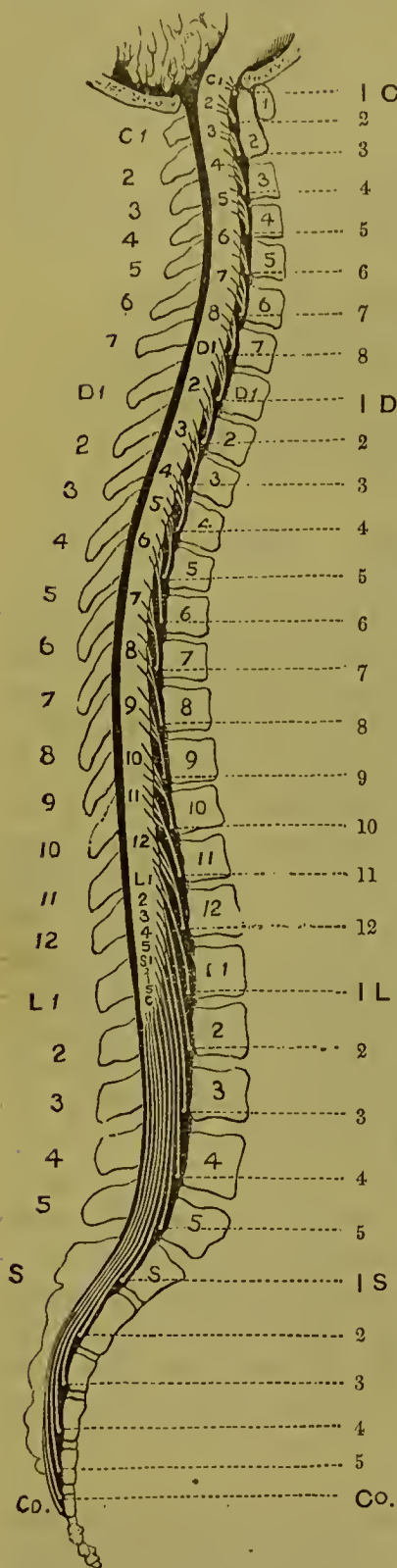


FIG. 56.

centre, and around this are concentric cloudy lines due to the irregular refraction of the white substance. Between the fibres is a peculiar

The posterior cornua isolate the posterior columns from the rest of the white substance. These posterior columns are divided by a median septum of connective tissue, and a little distance from this another incomplete septum (posterior intermediate septum), and corresponding to a depression on the surface, marks off a portion next the median septum, the "postero-median column," or "column of Goll," from the part next the posterior horn, "postero-external column," "column of Burdach," or "posterior root-zone." The latter name is given because the fibres of the posterior root pass through it. The rest of the white substance is divided, in the middle line in front, by the anterior median fissure, down which the pia mater and blood-vessels pass, and at the bottom of which is the anterior or white commissure. Between the anterior median fissure and the posterior cornu, the white substance is continuous and undivided, extending round the front and side of the cord. It is artificially divided into an anterior and lateral column, the line of division being the outermost of the anterior nerve-roots, which pass through the front of the cord, but there is no corresponding distinction of structure or function. The white substance varies in amount in different parts of the cord, and, as a whole, lessens gradually from above downwards (see Fig. 58). To this progressive diminution in size certain parts constitute exceptions.

The white substance is everywhere composed of medullated nerve-fibres, which vary considerably in size. In carmine-stained sections the axis-cylinder is seen within each fibre, not always in the

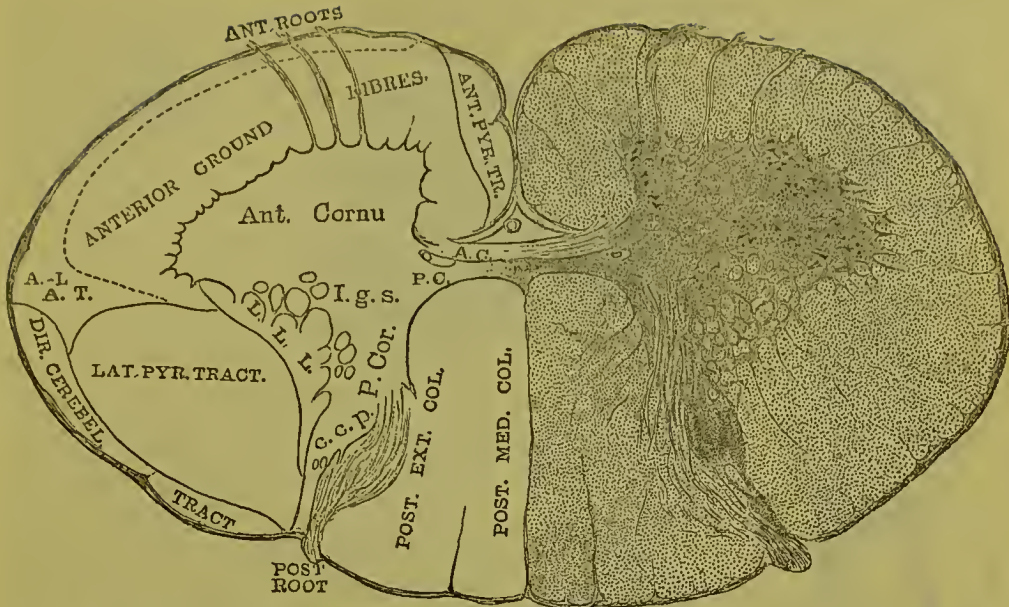


FIG. 57.—Diagram of a section of the spinal cord in the cervical region. A. C., anterior commissure; P. C., posterior commissure; I. g. s., intermediate grey substance; P. cor., posterior cornu; c. c. p., caput cornu posterioris; L. L. L., lateral limiting layer; A.-L. A. T., antero-lateral ascending tract, which extends along the periphery of the cord.

substance, the "neuroglia" or nerve-cement, the exact nature of which has been the subject of much investigation and discussion. It appears to consist of an albuminous matrix; embedded in this are fine fibres, which resist acetic acid, and these form a network. At their intersections are peculiar cells consisting of a nucleus and small cell body ("glia-cells," "cells of Deiters"). These were formerly regarded as stellate, and are still believed to be so by many authorities, the fibres being looked upon as their processes, but, according to Ranvier, they are flattened cells, without processes. The nerve-fibres, thus connected, lie in the meshes of a coarser network, formed by branching processes of finely fibrous connective tissue, proceeding from the pia mater on the outside, and from the grey matter on the inside. In these septa blood-vessels run, and some are occupied by nerve-fibres that have a horizontal course from one element of the cord to another. These tracts of connective tissue unite the pia mater so closely to the cord that the membrane cannot be readily detached, but in the intervals between the septa, the pia mater and cord are separated and connected by a very narrow layer, similar in structure to that which caps the head of the posterior horn, the so-called "gelatinous grey substance." This also continues the posterior horn to the surface of the cord (Fig. 57). In the dorsal region, where the head of the posterior horn proper is some distance from the surface, this tract of gelatinous substance is of considerable length. It fills up a narrow fissure, which extends inwards from the surface.

The *grey substance* varies in shape and size in different parts of the cord, being largest in the cervical and lumbar swellings, and corre-

sponding to the number and size of the nerve-roots given off. The

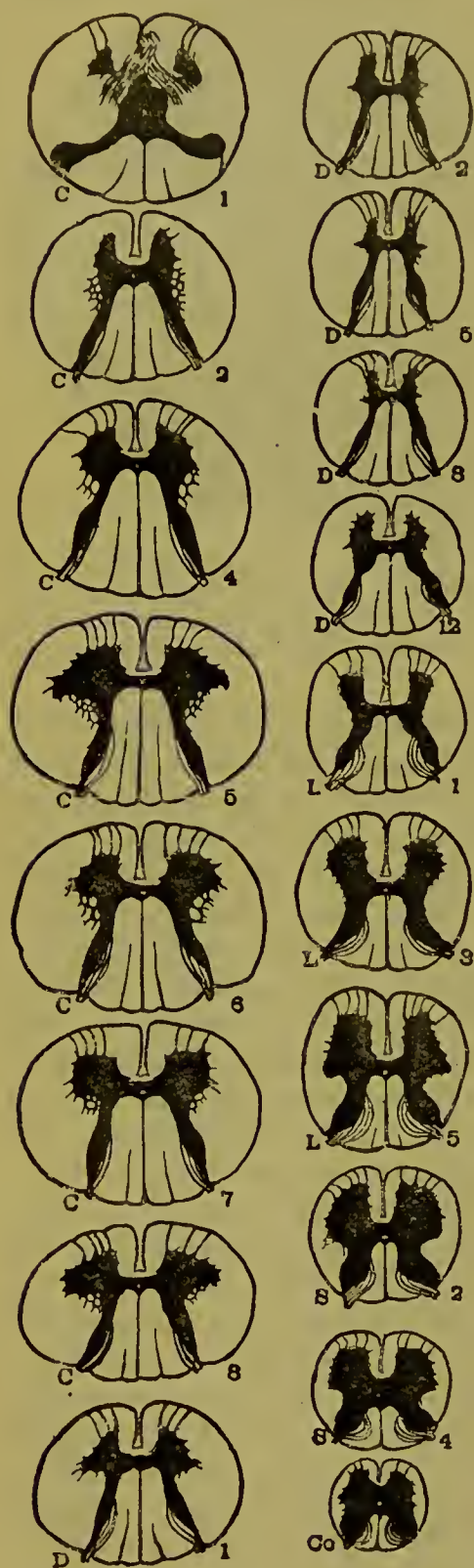


FIG. 58.—Diagram showing the relative size and shape of the cord and grey matter at different levels.

from the anterior commissure, white

variations in shape and size are indicated in the accompanying figure (58), and will be readily understood by an examination of this. Its division into anterior and posterior horns or cornua is familiar. The part on each side that intervenes between the two horns may be conveniently termed the "intermediate grey substance." It is customary to divide the grey matter into two varieties, "spongy" and "gelatinous." The latter forms a cap on the posterior horns, and is named on account of its naked-eye aspect. The spongy substance, which forms the rest of the two cornua, consists mainly of an excessively fine felty network of nerve-fibrillæ, with some cementing neuroglia and small cellular bodies, which vary in size and shape. Many are angular with processes, and are probably of nervous function. Through its course many larger nerve-fibres, medullated and non-medullated, and in it lie many nerve-cells of various sizes. Those in the anterior cornu are, for the most part, large "ganglion cells," nucleated, with many processes. One process has the aspect of the axis-cylinder of a nerve-fibre (Fig. 59), and can be sometimes traced into a fibre of an anterior root (Fig. 60). The other fibres divide and subdivide, their ramifications being lost in the sponge-like matrix of the grey substance. Nerve-fibres divide and join this ramification (Fig. 61). These cells lie in groups, between and through which course tracts of fibres, chiefly from the anterior roots. Other fibres come

FIG. 59.



FIG. 60.

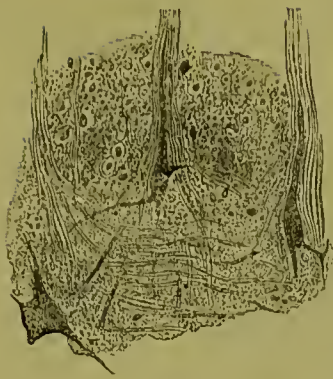


FIG. 61.



FIG. 59.—A nerve-cell from the anterior cornu of the spinal cord of man; *a*, unbranched process becoming the axis-cylinder of a nerve-fibre; *b*, pigment mass. (After Gerlach.)

FIG. 60.—Nerve-cells of the anterior cornu sending a process into the anterior root. (After Henle.)

FIG. 61.—A fine nerve-fibre dividing into two parts, each of which joins the plexus of fibrillæ formed by the branching processes of a nerve-cell. From the spinal cord of an ox. (After Gerlach.)

ganglion cells are certainly motor in nature, and give origin to at least most, and perhaps all, of the motor fibres of the spinal nerves. Birge, in a careful investigation, found that in each segment of the spinal cord of the frog the number of nerve-cells in the anterior cornu corresponds to the number of fibres of the anterior roots arising from that segment.

The arrangement of the nerve-cells of the anterior cornu is of some practical importance, on account of the frequency with which this part is the seat of limited lesions. They form, as already stated, certain groups, but these groups vary in different regions of the cord, partly under the influence of the shape of the horn, partly irrespective of this, and they even vary in parts of the cord that are near together. They are influenced by the course of the tracts of fibres of the anterior roots, which may pass through a group and break it up into smaller groups, although in a neighbouring section it is undivided (see Fig. 62, the cervical groups). Hence very different descriptions have been given of these groups, and the process of distinction has sometimes been carried too far. The groups that can most readily be recognised are the following:—In the inner anterior angle of the cornu is a small group, the *inner or medial group*. This is one of the smallest, and is absent in some parts of the cord, especially in the lumbar region. A much larger group lies near the anterior edge of the horn, in the middle, or a little to the outer side of the middle, the *anterior group*. External to this, in the outer extremity of the front part of the horn, is another group, the *antero-lateral group*. These two groups are often blended, so that the anterior group cannot be separately distinguished, as in the right hand mid-cervical figure. A third group, usually the largest, lies in the outermost part of the horn, behind its

front, usually in the posterior outer angle; it may extend inwards, half way across the horn. It is called the *external* or *postero-lateral group* (P.-L., Fig. 62). These three are the most important groups.



FIG. 62.—Diagrams of the groups of nerve-cells in the anterior cornu. Groups:—I., inner or medial; A., anterior; A.-L., antero-lateral; P.-L., postero-lateral; I. L. P., intermediate lateral process; P. V. C., posterior vesicular column or tract. The two mid-cervical sections are only a few millimetres apart, and show how the anterior group, separate in the one, may be blended with the antero-lateral group in a neighbouring part of the cord.

There is in some parts also a *middle group*, occupying nearly the centre of the horn. In the small cornu of the dorsal region often no well-defined groups can be made out, but when any can be recognised, they are generally the anterior and external.

Similar cells, usually smaller in size, and isolated, are scattered through the intermediate grey matter, and a group of cells of some size occupies a projection which extends outwards into the lateral column in the lower cervical and upper dorsal regions. It was termed by Lockhart Clarke the "*intermedio-lateral tract*," but (on account of other associations with the words "*lateral tract*") the projection is better designated the "*intermediate process*," and the cells the "*intermediate group*" (I.-L. P., Fig. 62). Isolated ganglion cells, medium and small in size, lie in the posterior horn. These are often fusiform, with many processes, and are supposed to be connected with some of the fibres of the posterior roots. They extend back into the caput cornu posterioris.

In the lower dorsal and upper lumbar cord, from the ninth dorsal to the third lumbar nerves, a group of nerve-cells lies in the inner part of the neck of the posterior horn, the "*posterior vesicular tract*" of Lockhart Clarke* (p. v. c., Fig. 62; see also Fig. 71). Most of these cells are fusiform in shape, but are placed vertically, so that they appear round or oval in transverse section. Some, however, present processes that run forwards or backwards. Nerve-fibres pass into and by it, which we shall consider subsequently. Although this tract is chiefly developed in the lower dorsal and upper lumbar cord, a few nerve-cells of similar character are met with in the same position in other parts, and are occasionally sufficiently numerous to form a small group.

The "gelatinous" grey matter which forms a cap on the posterior horn, *caput cornu posterioris* (c. c. p., Fig. 57) differs considerably in structure from the rest. It resembles closely the granule-layers of the retina in microscopical aspect, and apparently in chemical nature. It has been described as made up of granules, but, according to later researches, consists of a peculiar, translucent material forming an excessively fine network, which behaves to reagents like horny material. Numerous nerve-fibres pass through it, from the posterior roots, and these, coursing forwards, seem to divide it into columns, as it is viewed in transverse section. Although it is developed from the same embryological elements as the true nerve-structures, it has not, according to this view, a nervous function, but is to be regarded rather as a supporting structure, specially developed in connection with the sensory nerve-roots. Its extent and position are, it must be admitted, difficult to reconcile with this view of its nature. A few small ganglion cells lie in this gelatinous substance, chiefly on the inner side. Bundles of vertical fibres are also seen in transverse section, and these are often numerous just above the caput, especially on the inner side of the horn. Most of these are fibres of the posterior nerve-roots that have a vertical course for a short distance. In Fig. 71 the axis-cylinders of these fibres are seen to be changing from the horizontal to the vertical direction, and their relation to the posterior root-fibres can be distinctly traced. These fibres are sometimes so abundant that some of them probably belong in part to the postero-external columns (posterior ground-fibres). The gelatinous substance contains many vessels, running for the most part vertically.

White Substance.—We may now examine, in greater detail, the constitution of the white columns of the cord, and the probable course and function of the fibres they contain. The analysis has been greatly aided by two facts. The first (discovered by Türck) is that in certain tracts in the white substance the nerve-fibres undergo secondary degeneration when separated from their cells of origin, a degeneration analogous to that which, as we have already seen, occurs in the nerve-fibres

* Continental anatomists have done Lockhart Clarke the questionable honour of throwing aside the name he gave to this group and terming it "Clarke's column."

outside the cord. The process is analogous in general character, but we do not know that it is similar in histological nature. As we have seen, the degeneration in the peripheral nerve-fibres seems to vary in its character in different cases. Secondly, in the developing cord, the fibres of different tracts acquire their white substance at different

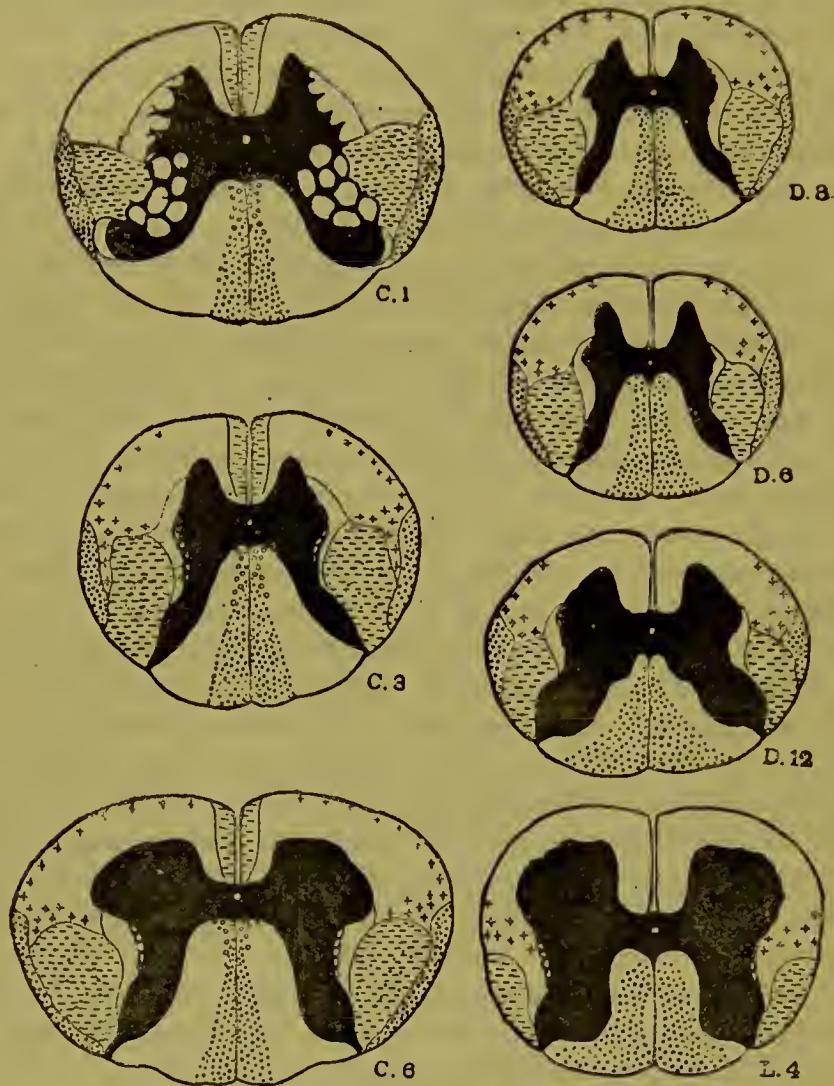


FIG. 63.—Diagram of the elements of the white substance at various levels of the spinal cord. (Modified from Flechsig.) The pyramidal tracts are shaded by short horizontal lines; the direct cerebellar tract by dots; the posterior median column by dots as it degenerates in disease of the lowest part of the cord or of the nerves of the cauda equina; the small circles in the forepart of this column indicate the area which degenerates in disease of the cervical enlargement. The antero-lateral ascending tract is shown by crosses. It is not represented in the first cervical section because there are no facts, at present, to show its exact position here; it probably lies chiefly in front of, and between, the direct cerebellar and pyramidal tracts, as c. 3. The anterior ground fibres, lateral limiting layer, and postero-external column are left white. Fig. 57 should be compared with this.

periods, and the study of these differences (by Flechsig especially) has not only confirmed, but also extended, the indications afforded by disease. The knowledge we possess is still incomplete. Some facts

ascertained have not yet found a practical application, but many others are of the utmost importance on account of the light they throw on the processes of disease.

It will facilitate the study of the subject if we consider first those tracts which undergo secondary degeneration, as our knowledge regarding these is the most complete. These degenerations are both ascending and descending. Only one set of fibres degenerates downwards, through a considerable extent,—those that continue through the cord the anterior pyramids of the medulla. They commence still higher, in the motor region of the cortex of the cerebral hemisphere, and are the conductors of voluntary impulses. From their relation to the anterior pyramids they are termed the *pyramidal tracts*. They are shaded by short horizontal lines in Fig. 63; compare also Fig. 57.

At the decussation of the pyramids about three quarters of the fibres usually cross to the other side. These pass down the cord in the lateral column, and constitute the *lateral* (or *crossed*) *pyramidal tract*. Those which do not decussate, pass down their side of the cord, in the inner part of the anterior column, at the side of the anterior median fissure, and constitute the *anterior* (or *direct*) *pyramidal tract*. Flechsig has found that the decussation is subject to many variations. In the majority of cases, the decussating fibres are between 70 and 80 per cent. of the whole. Sometimes they constitute a still larger proportion, and in one case (of 60 examined) all the fibres crossed. Sometimes fewer fibres decussate, only one half or even less than half; in one case 35 per cent., in another only 10. No case has yet been met with in which no fibres crossed.

The *lateral pyramidal tract* occupies the posterior half of the lateral column, outside the posterior cornu. It extends down to the end of the cord, even when it is originally small, and in consequence most pyramidal fibres do not decussate. Hence the fibres that most constantly decussate at the medulla are those for the leg. Through the greater part of the cervical and dorsal regions, this tract is separated from the surface by a narrow layer of fibres, the "*direct cerebellar tract*." In the upper part of the cervical region (third cervical segment), this tract lies farther forwards, so that the pyramidal tract comes up to the surface close to the posterior cornu (Fig. 63, c. 3), and here, if the tract is small, there may be a depression on the surface. In the lower part of the dorsal cord there is a similar movement forwards of the cerebellar tract, so that the pyramidal tract again comes in contact with the surface posteriorly (Fig. 63, d. 12), and as the cerebellar tract ceases at the last dorsal nerve, the pyramidal tract extends up to the surface throughout the lumbar enlargement (Fig. 63, l. 4). The inner side of the tract is in contact with the hinder part of the posterior cornu, near the surface, throughout the entire length of the cord, but further forwards it is separated from the base of the cornu and intermediate grey substance by a layer of nerve-fibres, termed by Flechsig the *lateral limiting layer* (L. L. L., Fig. 57). Among the fibres of the

pyramidal tract, in the dorsal and cervical regions, are scattered a few other fibres that belong chiefly to the cerebellar tract. The lateral pyramidal tract diminishes in size from above downwards. Its fibres curve inwards along the septa that extend from the grey matter into the lateral column, and they enter the grey matter between the anterior and posterior cornua. They pass inwards and forwards in the anterior cornu, and are then lost in the complicated structure of the grey matter. Their probable termination will be considered presently.

The *anterior or direct pyramidal tract* (called also the "column of Türk") descends the cord in the inner part of the anterior column, adjacent to the anterior median fissure. Its exact form and size vary; usually it bounds the fissure on each side; sometimes it forms only a small tract at the posterior part of the fissure; when large, it not only reaches forward to the front of the cord, but causes a prominence on the surface bounded by a distinct sulcus. Its extent down the cord, always less than that of the lateral tract, varies according to its



FIG. 64.—Ascending and descending degenerations from myelitis of the mid-dorsal region: the upper section is from the lower cervical, and shows the ascending degeneration of the post.-median column spreading out against the commissure. The other figure is from the lower dorsal, and shows degeneration of the pyramidal tracts, anterior and lateral.

size. It steadily lessens in area, and, if originally small, may not extend beyond the middle of the cervical enlargement; if originally large, it may be traced as far as the lumbar enlargement (Fig. 64); when bearing the usual proportion to the lateral column, it ceases about the middle of the dorsal region. Hence it seems that this tract contains chiefly fibres for the arm, a conclusion which harmonises with the fact that the lateral tract, even when small in the cervical region, extends down to the lumbar enlargement. But microscopical examination, even of the developing cord, throws no light on the way in which the fibres of this tract end. Flechsig could find no indication of their passage into the anterior commissure; at the same time his observations do not negative such an ending, and it is certain that many fibres of the anterior

commissure come from anterior columns and from the neighbourhood of this tract. The probability that the fibres decussate in the cord is strong, because most of them seem destined for the arm, and the relation of the arm is almost exclusively to the hemisphere of the opposite side. It is therefore probable that most of the fibres pass through the anterior commissure to the opposite grey matter.

Disease of the brain destroying the motor cortex, or the fibres which descend from it to the pyramids, causes descending degeneration,

limited to the related pyramidal tracts, anterior on the same side, lateral on the opposite side (Fig. 65). But in some cases slight degeneration has been also found in the lateral tract on the same side, extending into the lumbar region. Hence it is probable that some fibres of each anterior pyramid find their way to the lateral tract on the same side, and descend to the corresponding leg. This degeneration of the lateral tract on the same side as the lesion, is very marked just below the decussation of the pyramids, and therefore cannot be due to any lower decussation in the cord.* We shall afterwards see that there is other evidence that each hemisphere of the brain is connected with both legs, although chiefly with that of the opposite side.

How do the fibres of the pyramidal tract end? They seem all to pass into the intermediate grey matter, and forwards into the anterior cornu, among the motor nerve-cells. There they are lost in the plexus of nerve-fibres. There is the strongest indirect evidence that they divide and subdivide, and that their ramifications join the plexus of nerve fibrillæ constituted by the dividing processes of the ganglion cells. The evidence of this is twofold. As far as is at present known, only one process of a ganglion cell becomes an axis-cylinder; the others divide. The axis-cylinder process passes into an anterior root. It can be seen to do so in the case of some ganglion cells (Fig. 60), and, as we have seen, the number of ganglion cells and anterior root-fibres is the same in the frog, and therefore, presumably, in man. If so, since the pyramidal

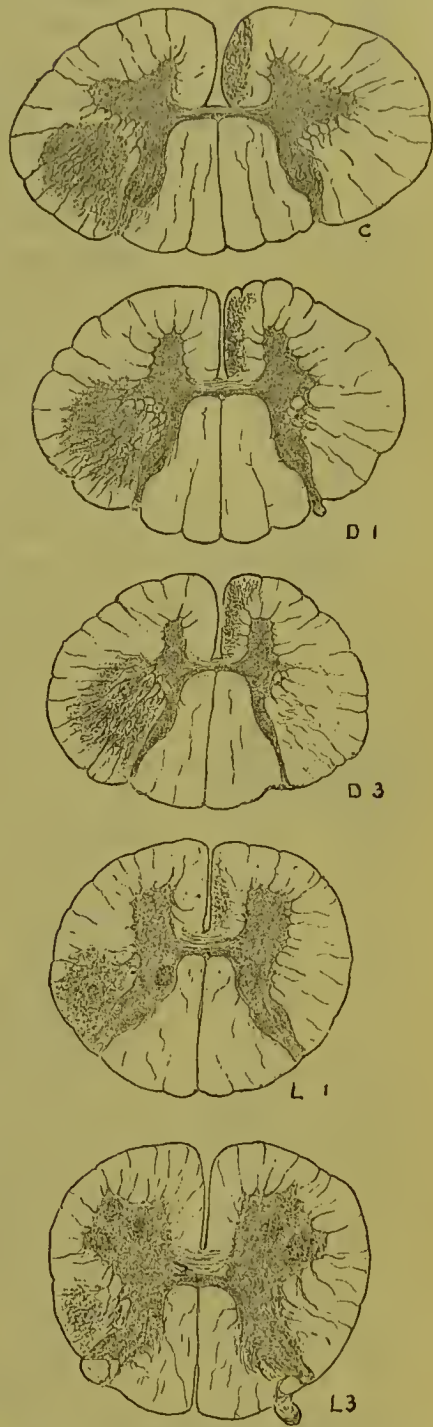
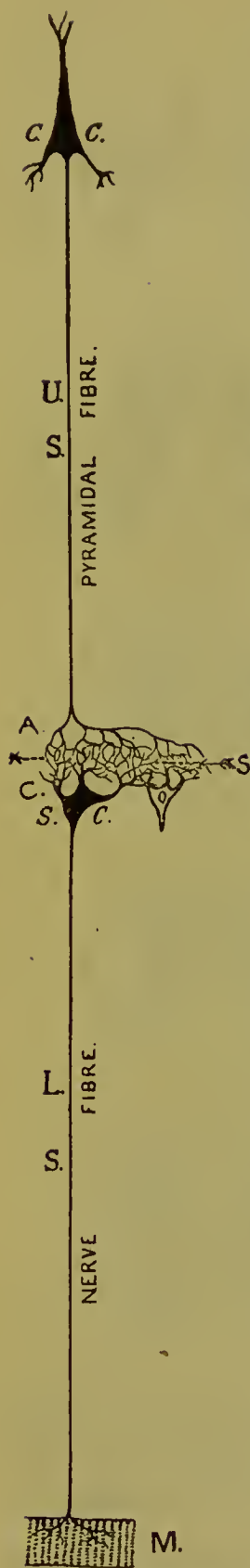


FIG. 65.—Descending degeneration of the pyramidal tracts in a case of hemiplegia from disease of the right cerebral hemisphere. (From sections prepared by Dr. Mott.)

* Pitres, 'Arch. de Physiologic,' 1874. Haddon and Sherrington, 'Brain,' Jan., 1886. An illustration of this bilateral degeneration will be found in the section on Diseases of the Brain.



fibres are certainly connected with the ganglion cells, the connection can only be by the ramifying processes of the cells and this involves a division of the nerve-fibre. The dividing fibre isolated by Gerlach, and shown in Fig. 61, may have been a branch of a pyramidal fibre. The second point in evidence of division is the fact that the nerve-cells and root-fibres are many times more numerous than the fibres of the pyramidal tract. But all (or at least most) of the motor root-fibres may be stimulated through the pyramidal fibres, and therefore the latter must be connected with all (or most) of the ganglion cells. This also involves a terminal ramification.

It is worth while to consider for a moment the whole motor path, from the cortex of the brain to the muscles. We may consider it as composed of two segments, an upper and lower (Fig. 66). Each consists of a ganglion cell above, a nerve-fibre, and the terminal ramification of the latter. The upper, "cerebro-spinal," segment consists of the cortical ganglion cell, and the pyramidal fibre which proceeds from the cell, passes through the brain and cord, and ends by dividing in the spongy substance of the anterior cornu. The lower, "spino-muscular," segment consists of the spinal ganglion cell, and the fibre proceeding from this, passing through the anterior root and nerve-trunk to the muscle, where it divides and ramifies on the muscular fibre. The elements of the two segments do not probably correspond in number. Each cortical-spinal element is connected with many spino-muscular segments.* It will be found that this conception of the motor path conduces to clearer ideas of many phenomena of disease.

Fibres degenerate upwards in both the lateral and the posterior columns. We may consider the latter first.

The *posterior column*, as we have seen, is divided into two parts by the imperfect "intermediate

* The terminal ramification of the lower segment is a complex "end organ," containing minute cells, and it is possible that the termination of the upper segment is also an analogous structure, containing cellular elements.

FIG. 66.—Diagram of an element of the motor path. U. S., upper segment; L. S., lower segment; C. C., cortical cell; S. C., spinal cell; A. C., anterior cornu; M., muscle; the dotted line indicates the junction of the two segments; S, path from sensory nerve-roots.

septum," a narrow "postero-median column," and a wide "postero-external column" (Fig. 57). Secondary degeneration in part confirms this distinction, but shows, at the same time, that the distinction must not be made the ground for a division of the two. If the posterior columns are interrupted anywhere in their course, ascending degeneration results, and this, a short distance above the lesion, is confined to the posterior median column (Figs. 64, 67, 68), in which the degeneration continues upwards to the medulla oblongata, and ends there in the grey matter of the "post.-pyramidal nucleus." The median columns degenerate in the same way when the lesion is not in the cord but in the pos-

terior nerve-roots of the cauda equina (Fig. 68). Hence, it is clear that these columns contain long fibres that are continuous with some of the fibres of the posterior nerve-roots.* The contrast between these degenerated columns, and the undegenerated post.-

* This inference has been recently contested by Betcherew on the ground that secondary degeneration sometimes passes nerve-cells. But in the central nervous system of man, as a general rule, the degeneration ceases where cells interrupt the fibres. This fact is so constant in so many parts that continuity of degeneration must be held as strong presumptive evidence of continuity of fibre, until the interruption has been demonstrated in the individual case.

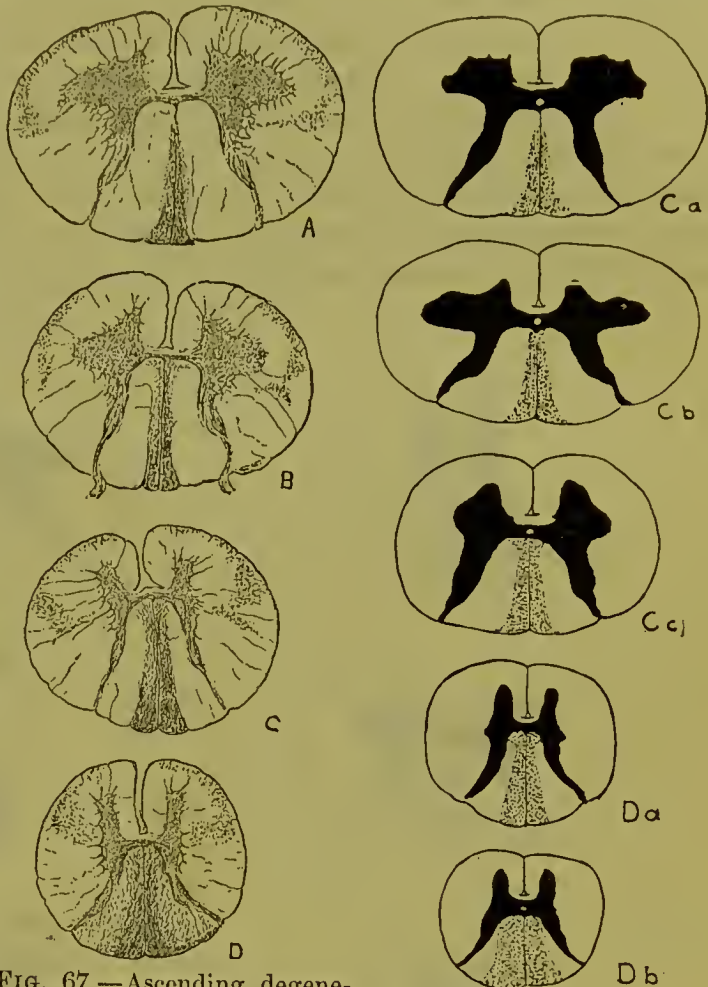


FIG. 67.—Ascending degeneration in the postero-median column and antero-lateral ascending tract. The cord was crushed at the first lumbar segment.

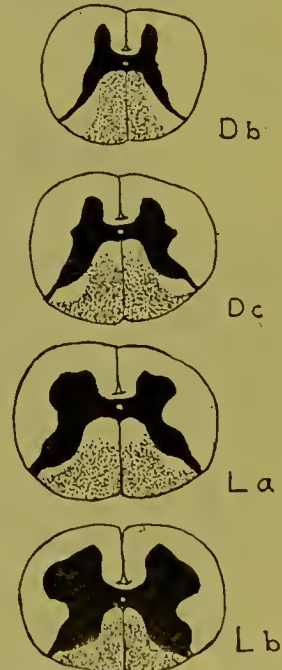


FIG. 68.—Degeneration after injury to the cauda equina. Ca, b, c, upper middle and lower cervical; Da, upper; b, c, lower dorsal; La, upper; and Lb, middle lumbar region. (After Schultze.)

external eolumns, through the greater part of the cord above the lesion (Fig. 67) is very striking, and justifies the distinction between the two. But just above the lesion the degeneration is not confined to the median eolumns; it spreads out into the post.-external eolumns, especially towards the posterior surface, and the more extensively, the nearer to the lesion, until close above this the whole extent of the posterior eolumns is diseased, except a small area close to the posterior eornu, sometimes only in the anterior part. This is true whatever be the seat of the lesion, whether it be in the cervical (Fig. 69), dorsal, or lumbar (Fig. 70) parts of the eord, and if

FIG. 69.

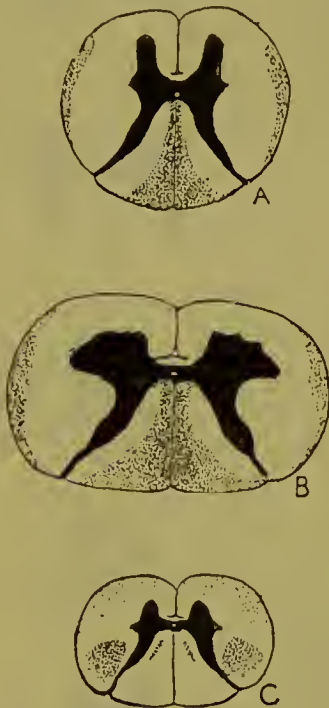


FIG. 70.

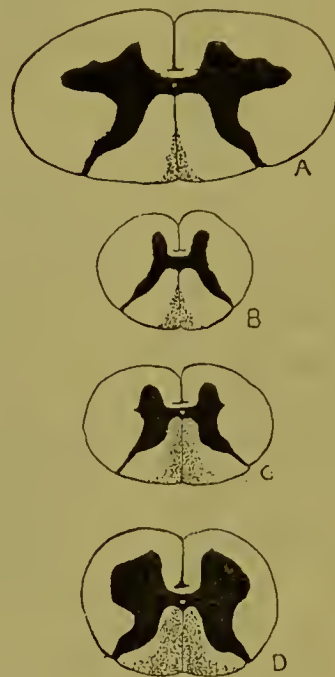


FIG. 69.—Degenerations after a lesion of the cord in the lower part of the cervical region. A, highest cervical; B, middle cervical $1\frac{1}{2}$ cm. above lesion. Degeneration of the posterior median eolumns, spreading slightly in A and widely in B into the post. ext. col. The circumferential degeneration is probably composed of the direct cerebellar tract and the antero-lateral ascending tract. C, upper dorsal 2 cm. below lesion. Degeneration of the lateral pyramidal tracts; "comma-shaped" degeneration in front of the post. ext. col.; slight degeneration of the anterior ground fibres.

FIG. 70.—Ascending degeneration after injury to the lowest part of the spinal cord and sciatic nerve-roots arising from it. A, cervical; B, lower dorsal; C, junction of dorsal and lumbar regions; D, middle of lumbar enlargement. (After Schultze.)

it is in the cauda equina, the degeneration spreads out in the same way in the lumbar enlargement (Fig. 68). This seems to show that all the way up the cord, fibres pass to the post.-median column from the post.-external eolumn, and that these are fibres from the posterior roots is highly probable in the lower part of the eord, and therefore probable also in all parts. As the fibres ascend the eord in the median column, they come to lie posteriorly in proportion as their source is lower. Those from the sciatic nerves, for instance,

in the lower cervical cord, only occupy the posterior half of the columns (Fig. 70). Those from the whole lumbar enlargement, or whole cauda equina (Fig. 68) reach forward to the commissure in the lower cervical region, spreading out a little near the commissure, where, however, the degeneration is much less dense than it is behind. Above the middle of the cervical enlargement, the degeneration from a lesion in the dorsal or lumbar regions, does not extend forwards beyond the junction of the anterior and middle third of these columns, but if the lesion is in the cervical region, the degeneration extends up to the commissure, even at the level of the second cervical segment (Figs. 69, A, 64), showing that the fibres from the cervical roots occupy the anterior portion of these columns.

The root-fibres that pass to the post.-median column through the hinder part of the post.-external column apparently do not decussate. This is not, however, the only source of fibres to the post.-median column. Fibres pass to it (1) from the neck of the posterior horn, across the anterior part of the post.-external column, curving backwards (Fig. 71, *x*); many of them extend almost to the postero-median septum. They come from the layer of fibres that lies on the inner part of the neck of the cornu, on the inner side of the post.-vesicular column, where this exists. Some of them may be distinctly seen to turn backwards in the neck as if they come either from the posterior roots or from cells near the caput cornu. But it is possible that some of them may be fibres from the post. commissure. (2) From the posterior commissure in the middle line. These are very numerous and conspicuous in the lumbar enlargement. They course backwards in the septum and then pass outwards on each side into the adjacent column. Flechsig believes that these are posterior root-fibres. They may easily decussate in the septum, but such decussation has not been proved.

In the lower lumbar region there is a narrow tract of fibres close to the posterior median septum, that is apparently of different nature from the rest of the column. It is lenticular on transverse section and is indicated by the clear area in L. 4, Fig. 63, and faintly in L b, Fig. 68. It is distinguished by a difference in time of development (Flechsig) and by freedom from the secondary degeneration that involves the rest of the column.

It is very doubtful whether all the fibres that enter the postero-median columns continue in these to the brain. Some certainly do, but the upward increase in size of the columns seems to be far too small for the accommodation of all the fibres that seem to pass to them. At the same time we have, at present, no indication of the mode in which fibres leave these columns.

At the medulla oblongata this column becomes filled with nerve-cells, the "*post.-pyramidal nucleus*," so called because the highest portion of this column has been termed the *posterior pyramid* of the medulla. The discovery that the fibres of this column are continuous with the nerve-roots invests the post.-pyramidal nucleus with considerable

importance since its nerve-cells are the first with which these root-fibres are connected. The upward degeneration of the post.-median fibres implies upward conduction; but their precise function is still uncertain. It will be further considered in connection with the localization of function in the cord.

The postero-external column ("column of Burdach," "posterior root-zone," Charcot) consists chiefly of vertical fibres. Many of the posterior root-fibres pass through it, horizontally or obliquely



FIG. 71.—Posterior cornu and column at the last dorsal segment. P. M. C., postero-median column; P. E. C., postero-external column; P. M. S., posterior median septum; P. C., posterior commissure; *v*, commissure vein. P. V. C., posterior vesicular column; C. C., caput cornu; P. R., posterior root; *a*, an artery; *d, d, d*, adjacent to a strip of the lateral column, indicate the tracts of fibres passing from the vicinity and interior of the posterior vesicular column along the septa of the lateral column, to form the direct cerebellar tract; *x, x*, tracts of fibres passing from the neck of the horn, near the post. vesic. col., to the post.-median column.

(Fig. 57). Most of these curve inwards to the posterior horn, but the secondary degeneration just described seems to show that some must also pass obliquely upwards and inwards to the median column. Across the anterior part fibres also pass, as just described, between the neck of the posterior horn and the median column. The other vertical fibres of the external column, "posterior ground fibres" of Fleehsig, have apparently only a short course, since they only degenerate for a few centimetres above or below a lesion. The longest descending degeneration is of a comma-shaped bundle, in the middle of the anterior third of the column in the dorsal region, which degenerates downwards for four or five centimetres (Fig. 69). These vertical fibres, of short course, may connect the grey matter of the posterior horn at different (but adjacent) levels. This column is larger in the enlargements than it is in the dorsal region, partly in consequence of

the larger number of root-fibres in the former. Above, it also ends in a grey nucleus.

The function of this column is still unknown, although much interest attaches to it as the seat of the special central lesion in locomotor ataxy. How far the inco-ordination of this disease depends on damage to the root-fibres that pass through the column, or to fibres that connect the grey matter at different levels, is still unknown. We shall return to the question when discussing the disease.

The "*direct cerebellar tract*," is another column of fibres which degenerate upwards. It forms a layer at the periphery of the lateral column, outside the pyramidal tract (Fig. 57), but does not extend through the whole length of the cord, ceasing at the level of the first lumbar nerve (Fig. 63). The anterior part of the tract (as seen in section) does not extend forwards beyond the anterior level of the lateral pyramidal tract, but, as we shall presently see, there are other fibres in front of it that also degenerate upwards, and these have given rise to an erroneous opinion that the cerebellar tract extends farther forwards than it actually does. But at the level of the second cervical nerves, and also in the lowest part of the dorsal region, the tract lies a little anterior to its position in the rest of the cord, and hence at these places, as we have seen, the pyramidal tract comes to the surface behind the cerebellar tract, close to the posterior nerve-roots, up to which elsewhere, the cerebellar tract extends. The tract increases somewhat in size from below upwards, and hence receives fibres throughout its course, but the chief bulk of its constituent fibres enter it at the level of the lowest dorsal and first lumbar nerves, *i. e.* at its lowest part. These fibres come from the grey substance, coursing to it through the lateral column, along the septa that cross the pyramidal tract from the grey matter. In the latter these fibres are conspicuous objects at this level (Fig. 71), passing transversely and obliquely from the front of the posterior vesicular tract. Into this many of them can be traced; others change their direction and become vertical, probably passing into the vesicular tract at a different level. It is probable that the fibres come from the tract; the cerebellar tract is chiefly formed at that part of the cord at which the posterior vesicular column is chiefly developed, and the cells of this column have been found atrophied when the cerebellar tract is degenerated.* Besides the fibres that are gathered into the compact tract, others, belonging to its system, ascend among those of the adjacent lateral pyramidal tract.

The cerebellar tract only degenerates when a lesion of the cord is at, or above, its place of origin, the junction of the dorsal and lumbar regions. If the lesion is in the lumbar enlargement, as in the case shown in Fig. 67, the tract does not degenerate. At the level of the first cervical nerve (where the pyramidal tract leaves the lateral column to cross into the anterior pyramid of the medulla) the

* *E. g.* by Minkowski, 'Deut. Arch. f. kl. Med.,' Bd. xxxiv, p. 433. Slight degeneration of the pyramidal tracts was the only other lesion.

cerebellar tract lies in front of the "grey substance of Rolando," (formed from the caput cornu posterioris) and passes up, in the restiform body, to the cerebellum. We have no definite knowledge of its function. It certainly conducts upwards, and Flechsig thinks it may conduct impressions from the muscles of the trunk.

We have seen that the lateral pyramidal tract, although in contact, behind, with the head of the posterior cornu, does not extend quite up to the neck of the horn or to the intermediate grey substance, the two being separated by "*lateral limiting layer*" of vertical fibres, in part broken up by processes from the grey matter. In the upper cervical cord, and also to a less extent in the dorsal cord, this layer extends forwards outside the anterior horn. It consists of fine fibres that pass into the grey matter, forming curves, some with the convexity downwards, others with the convexity upwards. This course, and the fact that the tract does not degenerate through any considerable extent, suggests that its fibres have but a short course, and connect the grey matter at different levels.

The rest of the lateral column in front of the pyramidal tract is termed by Flechsig the *anterior mixed lateral zone*, because its fibres vary in size, course, and date of development. Secondary degeneration, however, as I pointed out some years ago,* enables us to distinguish a band of fibres lying immediately in front of the pyramidal and



FIG. 72.—Cervical section. Ascending degeneration in post.-med. col. and ant.-lat. ascending tract, secondary to a lesion in the dorsal region. (From a section prepared by Dr. Mott.)

cerebellar tracts, which degenerates upwards throughout the cord. The zone extends across the lateral column, as a band which is largest in area near the periphery of the cord, where it fills up the angle between the pyramidal and cerebellar tracts, and it reaches the surface of the cord in front of the latter tract; it then extends forward in the periphery of the anterior column, almost to the anterior median fissure. It may be conveniently termed the *antero-lateral ascending tract*. (Figs. 57; 63, crosses; and 67). It has often

been confounded with the cerebellar tract, and it is this which has given rise to the impression that the cerebellar tract extends farther forwards than it really does. In the lumbar region, the tract lies across the lateral column on a level with the posterior commissure, from which its fibres probably come (Fig. 63, L. 4). In the upper cervical (ib, c. 3) it extends also between the pyramidal and direct cerebellar tract, and probably occupies a similar position at the level of the first cervical. This tract has recently been found by Betcherew to undergo development at a different period from the rest of the lateral column. It is almost certainly a sensory tract, and physiological facts seem to show that its fibres are connected with the posterior roots of the

* 'Diagnosis of Diseases of the Spinal Cord,' first ed., 1879.

opposite side. Fibres probably pass to it through the whole length of the cord and these are mingled together, so that the degeneration from a lesion of the lower part of the cord is not dense.*

Some fibres of the anterior part of the lateral column are large in size, and curve upwards and downwards into the anterior horn. They must be of short course, and may be fibres of the anterior nerve-roots, which ascend and descend to nerve-cells on a different level from that of the roots by which they leave the cord.

The fibres of the anterior column, excluding the anterior pyramidal tract, are termed by Flechsig the *anterior ground-fibres* (Fig. 57). They are not separable, structurally or by development, from those of the lateral limiting layer, and it is doubtful how far the distinction of the two is warranted. The ground-fibres do not degenerate through any considerable extent of the cord, and some probably connect the anterior cornua at different levels. Moreover, some of the fibres pass to the anterior commissure, and thus, by the agency of these columns, a connection may be established between the two anterior cornua at different levels.

As far as investigations have hitherto gone, the direction in which fibres degenerate, in the central nervous system, is the direction in which they conduct. We may therefore feel reasonably sure that the fibres that degenerate downwards from the brain, or upwards to the brain, conduct as they degenerate, and through the same extent. But we cannot affirm that the tracts which do not degenerate through any considerable extent, do not form part of conducting paths to or from the brain. All we can feel sure of is that they do not constitute a continuous conducting path, such as the pyramidal tracts and the tracts that present a continuous ascending degeneration.

The *commissure* of the spinal cord lies between the bottom of the anterior fissure and the posterior columns, and in it alone is there a passage of fibres from one half of the cord to the other. It consists of two parts, an anterior or white, and a posterior or grey commissure. The *white commissure* varies in thickness in different parts of the cord, and is largest in the lumbar region. It is composed of medullated fibres, which cross in the commissure in such a manner that the anterior fibres on each side pass out in the posterior part of the commissure on the other side. The fibres in front pass into the anterior white column, those behind into the grey substance; hence they appear to establish a connection between the anterior column of one side and the anterior cornu of the other. Whether they go to ganglion cells or to the anterior nerve-roots is not known. It is not likely that any considerable number of fibres have the latter termination, but some are thought to pass from the roots of one side to the inner group of nerve-cells of the opposite cornu (Laura and Pick).

* Other illustrations of the degeneration of this tract will be found in the section on Locomotor Ataxy. For an example of its degeneration and references to previous observations, see Tooth, 'St. Bartholomew's Hospital Reports,' 1885, p. 137.

Some fibres, moreover, pass across the intermediate grey substance, and, reaching the lateral column, assume a vertical direction. It is probable that some fibres are decussating fibres of the direct pyramidal tract, so that part of the anterior commissure is to be regarded as a continuation of the decussation of the pyramids. Lastly, a few fibres of the anterior commissure have been thought to come from the posterior nerve-roots.

The *grey commissure* contains very fine medullated nerve-fibres, a large number of which pass backwards in the posterior horn of each side, and appear to be continuous with the fibres of the posterior roots. It is highly probable that, by this means, a considerable number of the posterior root-fibres cross (or fibres from cells in which the root-fibres end), and that thus the partial decussation of the sensory path in the cord is effected, which is known to take place a short distance above the entrance of the nerves. If the grey commissure consisted only of root-fibres, it has been estimated that it could contain only about one half of those of the posterior roots. Some of the fibres that thus cross doubtless find their way to the antero-lateral ascending tract in the opposite lateral column. Others leave the commissure in the middle line, and pass backwards to the posterior median column, but whether these decussate or not we do not know.

Within the grey commissure is the central canal of the spinal cord, lined with epithelium, and surrounded by granular material, similar in nature to that which caps the posterior horn. It is often filled up in adult cords.

The *course of the root-fibres* remains to be considered. The anterior roots pass through the anterior columns in bundles which are distributed through an area of the column approximately corresponding to the width of the anterior surface of the cornu. In the grey substance, fibres from most of these bundles pass in three different directions, outwards, inwards, and directly backwards in the middle of the horn, and thus cross each other and the fibres from the anterior commissure. Many of the fibres enter the groups of nerve-cells and certainly end in these cells. Some of those that pass outwards, enter the lateral white column ("mixed zone") and probably, after a short course in this, re-enter the grey matter at a higher or lower level. Some of the fibres that pass inwards may pass through the commissure to the opposite anterior horn.

The *posterior root-fibres*, although arranged in a continuous vertical series at their surface attachment, divide into two sets, one of which passes through the "*caput cornu posterioris*" (Fig. 71), the other through the adjacent part of the post.-external column (Fig. 57). The former course through the gelatinous substance, sometimes in a compact bundle (Fig. 71), sometimes in curves of which the concavity is turned towards the centre of the head. Some of the fibres bend into a longitudinal direction above the caput (Fig. 71) and are seen in section

as rounded bundles. After a short vertical course, upwards or downwards, these fibres again pass forwards into the horn. Of the fibres that pass through the postero-external column many pass inwards and enter the posterior cornu in front of its head. These have not all a horizontal course; some pass obliquely upwards or downwards. Some may even have a vertical direction for a short distance. It is not likely that any fibres of the posterior roots pass far upwards in the postero-external column, but it is probable that some pass inwards, through this column, to the postero-median column, since the secondary degeneration of the posterior median column extends, below, into the outer column, when it is due to disease of the roots outside the cord.

In the posterior horn, many fibres of each set pass to the posterior commissure; others are believed to go to the posterior vesicular tract (where this exists) while some pass forwards into the anterior cornu, chiefly towards the outer group of nerve-cells, and a few pass towards the anterior commissure. It is probable that the fibres that pass to the posterior vesicular tract are connected with its cells, whether directly or indirectly through their ramifying processes and the spongy plexus, we do not know. Those that pass towards the posterior commissure are probably interrupted by nerve-cells in the posterior cornu.

BLOOD-SUPPLY TO THE SPINAL CORD.—The arterial supply is constituted by small branches, derived from the vertebrales, intercostals, and other arteries, which reach the cord by the anterior and posterior roots. The anterior pass for the most part inwards, to the anterior median fissure, where they are connected by vertical branches, continuous in direction, which are described as the “anterior spinal artery.” Branches also pass backwards into the anterior median fissure, at the bottom of which they divide, and the small arteries enter the cord, passing outwards through the anterior commissure, and again dividing in the grey substance. Small branches reach the lateral column (Ross). The fibres of the anterior commissure are displaced by the vessels, and hence, in section, the commissure often appears to be interrupted. The posterior spinal arteries give branches to the *caput cornu posterioris*, which course upwards and downwards in it (*a*, Fig. 71), and then divide into small branches which extend outwards and inwards, ramifying in the pia mater. From this ramification, arterioles pass inwards into the white substance along the connective-tissue septa, send off minute twigs among the nerve-fibres, and end in the grey substance. The branches into the lateral column do not reach the grey substance the branches in the grey substance extend into the inner part of the lateral column.

The *veins* of the cord pass in two directions. Some, especially those of the white substance, pass outwards along the septa, and those on the surface of the sides and back of the cord are gathered into a “posterior spinal vein.” Others, especially of the grey matter, pass inwards to a large vertical vein that lies in the grey commissure,

a little distance from the central canal on each side (*v.* Fig. 71, compare also Fig. 57), from which branches pass forwards to the anterior median fissure, and through this to an "anterior spinal vein." The anterior and posterior spinal veins deliver their blood, through communicating veins, into the huge venous plexuses that lie outside the dura mater, and which receive blood also from the bones, and from the structures and skin behind the spine. But the veins of the spinal cord cannot be injected from these plexuses, not because there are valves in the connecting branches, but apparently because they form so trifling a proportion of the total connections. From the plexuses blood passes to the various vertebral, cervical, and intercostal veins.*

Although there is a vertical continuity of the vessels of the cord, it is probable that the course of the circulation is, in the main, horizontal. From the very tortuous course of the path by which arterial blood enters the cord, it is evident that the pressure to which the arteries of the cord are exposed must be far lower than that in the arteries of the brain, and hence they are far less liable to degeneration and to rupture. On the other hand, the conditions that prevent an injection of the plexus of veins outside the dura mater from passing into the veins of the cord, must save the latter from the extreme overdistension to which they would otherwise be liable, when there is a hindrance to the return of blood from the plexus.

FUNCTIONS OF THE SPINAL CORD.

We are now in a position to consider the functions of the spinal cord, and the structures by which they are subserved. The functions are these:—(1) The conduction of motor nerve impulses from the brain and sensory impulses to it. (2) The spinal cord constitutes a series of centres for reflex action. (3) It contains certain centres that ultimately govern nervous action in structures under the immediate control of the sympathetic system of nerves, the bladder, rectum, blood-vessels, &c. (4) It influences the nutrition in all parts to which its nervous control extends.

MOTOR CONDUCTION.—We have already seen that the path of motor impulses is through the pyramidal fibres (in the anterior and lateral pyramidal tracts), the anterior cornua, and the anterior nerve-roots. The ganglion cells form part of the path, and so also probably does that part of the plexus of fibrillæ in the cornu which intervenes between the pyramidal fibres and the ganglion cells. We have also

* A fuller account of the vessels of the cord will be found in some papers by Dr. Ross, in 'Brain,' vol. iii, p. 80. At present we are not able to make a practical application of fuller details than are given in the text above.

seen that the path may be regarded as consisting of two segments, an upper, cerebro-spinal, and a lower spino-muscular (see p. 116). The conduction in the cord is chiefly on the side opposite to the hemisphere of the brain in which the impulses originate, to a less extent on the same side in the upper part of the cord (anterior pyramidal tract), the path crossing lower down by the anterior commissure. The fibres for the leg, however, probably pass in part down the lateral tract of the same side, establishing thus a connection between the leg and both hemispheres of the brain. This is probably true also of the path to the bilateral muscles, such as the intercostals, and to other muscles of the trunk, and likewise to those muscles that pass from the trunk to the arm, muscles that are often used with their fellows on the opposite side. None of these muscles are ever completely and permanently paralysed by a lesion on one side of the brain. We do not know whether this relation is subserved in any degree by fibres of the direct pyramidal tract.

The fibres of the anterior, motor, nerve-roots, all of which can probably be stimulated through the pyramidal tracts, are far more numerous than the fibres of these tracts. The latter apparently end in a complex mechanism, constituted by the motor nerve-cells and fibrillary network. This mechanism is such that the stimulation of certain pyramidal fibres excites to action a much greater number of nerve-cells, so connected and arranged as to produce, through the related nerve-roots, a complex movement in which many muscles may take part. The nerve-cells thus connected may not all be at precisely the same level, and only some of those at a given level may be thus associated. The simpler the movements, and the fewer their possible variations, the fewer pyramidal fibres may suffice for the production of the movements. Thus it is conceivable that for such movements as those of the intercostal muscles, which are simple and constant, very few pyramidal fibres may suffice, whereas a much larger number must be necessary for the highly variable movements of the hand.

REPRESENTATION OF MOVEMENTS IN THE SPINAL CORD.—It is important to inquire how far we can identify these various motor mechanisms of the anterior grey matter. There are several sources of information on this subject. We have already seen that the arrangement of fibres in the anterior roots is such as to associate certain movements with certain nerve-roots, and it is a reasonable assumption that this corresponds, to some extent at least, with the arrangement in the grey matter. The sources of our information on this point are the limited facts of anatomy, rare cases of restricted disease of the nerve-roots in man, and especially the experiments of Ferrier and Yeo on monkeys. We have already considered the indications that these experiments afford, and their suggestiveness, and we have seen that their value is relative rather than absolute. As regards the grey matter, we must remember also that there may not be a strict horizontal corre-

spondence between the nerve-roots and the nerve-cells, because it is probable that some root-fibres enter the antero-lateral white columns, and are connected with nerve-cells at a higher or lower level than that at which they leave the cord. Another source of information is the degeneration of nerve-cells that follows slowly on an amputation of a limb, and the condition of the spinal cord in congenital absence of part of a limb. The last and most important source of information is supplied by cases of limited disease of the anterior cornua, in which the position of the lesion and the distribution of the resulting palsy can be compared. Destruction of nerve-cells causes degeneration of the motor fibres proceeding from them, and wasting of the muscles to which those motor fibres proceed. This alone affords us certain knowledge. Cases of clear significance are, however, rare, and it will be long before our knowledge can be complete. Meanwhile, we may learn something of the central association of muscles by observing what muscles are most frequently paralysed together by such disease. This subject has been carefully studied by E. Remak* and many valuable isolated observations have been published. It is only the associations which are frequent that can be allowed significance, because it is not uncommon to have more than one focus of disease in the grey matter. The following summary presents the conclusions that seem probable from the evidence at present available.†

The various facts for the most part fully confirm the conclusion of Remak, corroborated by the experiments of Ferrier and Yeo, that most movements and muscles are represented in vertical tracts, and the whole anterior grey matter, at any one nerve-segment, contains cells that are concerned with different movements. An extensive lesion of small vertical extent may thus weaken many movements, but abolish none. The special representation of the muscular function, *i. e.*, of definite movements, is no doubt related to special groups of nerve-cells, but a single group may be concerned in more than one associated movement. Different groups are probably intimately connected, because we know that the contraction of any muscle is accompanied by a slighter but proportioned contraction of its antagonists. Anatomical connections in both spinal cord and brain doubtless underlie this association.

CERVICAL ENLARGEMENT.—We are chiefly concerned with the lower brachial part of this region, that below the fourth cervical. The upper part contains centres for the muscles of the neck, especially for the sterno-mastoid and upper part of the trapezius. The diaphragm is probably represented in the grey matter at the level of its nerve-roots, the fourth cervical. In the brachial region of the cervical enlargement, the muscles of the shoulder are represented chiefly in the upper part; the intrinsic muscles of the hand in the lower part. The flexors

* 'Archiv f. Psychiatrie,' vol. ix.

† A useful summary of the evidence up to the middle of 1884, was given by Dr. Allen Starr, in the 'American Journal of Neurology,' Aug. and Nov., 1884. The subject is a tempting one for theory, but it is very important to keep to the solid ground of facts, however limited it may be in extent.

of the elbow are represented above its extensors; and the supinators and extensors of the wrist above the flexors of the wrist.

Deltoid, scapular muscles, pectoralis, and serratus.—Fifth and sixth segments, for the most part the sixth, and probably the outer group of nerve-cells. The centres for the two parts of the pectoralis are separate, and are associated—the clavicular with that of the serratus magnus, the costal with that of the latissimus dorsi (see p. 26). These associated muscles are represented near together, but not at the same spot; the association is often present in disease of the anterior cornu, but one muscle may be affected without that which is commonly associated with it.

The *Flexors of the elbow* and *supinators* probably correspond nearly in level with the deltoid. The whole of this series of muscles may be affected alone by disease of the nerve-roots (see p. 77) or of the grey matter, and then we have the “upper arm type” of palsy, described first by Erb.

Extensor of the elbow (triceps): probably the middle of the brachial region, chiefly the seventh segment, extending down to the eighth, and perhaps up to the sixth, possibly chiefly in the outer group of nerve-cells.

The *Extensors of the wrist* are represented above those of the elbow; probably chiefly at the sixth or sixth and seventh segments, and not from the postero-external, but from the other groups. *Flexors of the wrist:* below the extensors, nearly on the same level as the extensors of the elbow, at the seventh and eighth segments, and probably also from one of the outer groups of cells. The *pronators* have nearly the same representation as the flexors of the wrist.

Long Extensors of the fingers: upper part of the brachial region, about the level of the sixth or between it and the seventh segment, probably from one of the anterior groups of cells; not from the postero-lateral group. *Long Flexors of the fingers:* below the extensors; probably seventh or eighth segments.

Intrinsic muscles of the hand: lowest part of the brachial region, and the thenar muscles a little higher than the interossei; the latter being represented in the first dorsal segment.* The intrinsic muscles are probably related less to the outer groups of cells than to the inner and anterior groups. We have seen that this is probably true also of the long extensors of the fingers. The two groups of muscles often act together, and their central connection was illustrated by a case of concussion-lesion of the cord under my care, in which the two outer interossei were paralysed and wasted, and also the part of the long extensor supplying these two fingers, but no other muscles.

LUMBAR ENLARGEMENT.—Our knowledge of the representation of muscles in the lumbar enlargement is very slight, and we have scarcely any definite facts concerning their relation to the cell-groups. The postero-external group preponderates over the others in size, even more than in the cervical cord, and doubtless also in importance. The few pathological observations of changes in the several groups are not altogether consistent. The chief facts available relate to the probable segmental level and association of the centres, and are as follow:

Cremaster, second lumbar segment. *Psoas*, second, *iliacus* third lumbar; the two muscles, one in function, are probably related to a group of cells extending through both segments. *Adductors*, fourth lumbar segment. *Gluteal muscles* (extensors of hip), fourth and fifth lumbar segments.

* This is the indication of the majority of cases. Nevertheless Sahli has recorded a case of atrophy of all the intrinsic muscles of the hand, in which the lesion did not extend below the seventh cervical: the eighth cervical and first dorsal segments were perfectly normal. I have seen a case of atrophy limited to the deltoid and intrinsic muscles of the hand, due to an acute process, probably hæmorrhage, but in this case there may have been two lesions.

Extensors of knee.—Third and fourth lumbar segments; probably from the same group as the flexors of the hip; the two sets of muscles are often affected together, and sometimes the adductors suffer with them. The *Sartorius* is probably related to the third segment, but not to the same cell-group as the extensors of the knee (to which indeed it does not belong; see p. 34). It usually escapes in atrophic palsy of the extensors. *Flexors of knee*, fifth lumbar and first dorsal segments. Their centre is thus below and certainly distinct from that of the extensors.

The muscles of the lower leg, moving the foot and toes, are related to the fourth and fifth lumbar and the first sacral segments. The *calf muscles*, the *tibialis anticus*, and the *peroneus longus* have certainly separate centres and are often affected separately. The *tibialis anticus* may suffer alone, or may alone escape when the other muscles in front of the leg are paralysed. The muscles of the calf may be affected when those in front of the leg are not. It is probable that the *peroneus longus* is the highest in central representation, and is related to the fourth lumbar segment.

The *intrinsic muscles of the foot*, especially the *interossei*, are the lowest in central relations, being connected with the second sacral segment. The position of their centres, in the lowest part of the lumbar enlargement, thus corresponds to that of the analogous muscles of the hand, in the lowest part of the cervical enlargement.

SENSORY CONDUCTION.—Our knowledge of the sensory path in the cord is far less definite than that of the motor path, in spite of the fact that it has been the subject of a large number of experimental investigations. The subject is a very difficult one for experimental solution, on account of the difficulty of ascertaining the condition of sensation in animals. The indications afforded by disease are equivocal for another reason. We have seen that, in the sensory nerves, a lesion which permanently interrupts motor conduction may scarcely affect sensory conduction, and the same thing is apparently true of the spinal cord. If loss of feeling results from an acute lesion, it often quickly passes away, although motor palsy remains complete and absolute, and this when all the elements of the cord seem equally damaged by the morbid process. In cases of chronic disease, such as compression, which also acts equally on all the structures, the same escape of sensory conduction is often observed. The result, in acute cases, has been thought to indicate that the sensation may find new paths, but this explanation can hardly apply to cases in which all the elements are equally compressed. It is more probable that the nerve-fibres suffer partial damage, without interruption of the axis-cylinder, so that their conducting power is lowered, and not lost, and suffices for sensation but not for motion. We have seen reason, on more than one ground, to believe that this is the case in partial lesions of the peripheral nerves (pp. 50 and 51). Hence we may find disease in those elements of the cord, to which other facts point as the sensory path, and yet there may have been no corresponding loss of sensation. In an area damaged by sclerosis or chronic myelitis, it is common to find many nerve-fibres retaining their form, although evidently changed in nature,

since their white substance stains more readily than in health; it is common also to find axis-cylinders surrounded by a sheath of myelin much narrower than normal, and this in parts in which there is a considerable increase of connective tissue. Such fibres may still possess some conducting power. Hence we cannot infer, because the supposed sensory tracts are visibly diseased in a case in which there was no loss of sensibility, that these tracts have not the function assigned to them, unless we can feel sure that all the axis-cylinders are destroyed. For this reason, and because morbid processes are often wide in distribution, the evidence that can be obtained by comparing the position of lesions with the sensory symptoms they produce, is limited and uncertain. Another source of information is the effect of partial transverse lesions of the cord, intense in degree, but limited in area, especially traumatic lesions affecting one half of the cord or part of one half. Cases of this kind are of the highest value, and it will probably be by these that the question will be ultimately decided. But the cases are few, at present, in which the extent of lesion and the range of symptoms have been accurately ascertained.

It would take too much space even to enumerate the various conclusions which have been reached by the many investigators who have endeavoured to solve the problem of sensory conduction. It may suffice, for practical purposes, to point out the evidence that seems most important, and the conclusions that seem, on the whole, most probable. The earlier experiments of Brown-Séquard established one important fact, which has been fully confirmed by pathology, that the chief part of the sensory path crosses the middle line soon after it enters the cord, and passes up to the brain on the other side. These experiments also suggested that one upward path is in the intermediate grey substance. This possibility cannot be said to be entirely disproved, but later investigations have given it no confirmation, and it is on the whole improbable. The experiments by others which seem to deserve the greatest weight are those of Schiff and Woroschiloff. The latter employed, as a test of upward conduction, the effect of sensory stimulation on the vessels. These experiments, taken together, point to two regions of the white substance as containing the sensory path: the posterior columns and the lateral columns. In the lateral columns the path cannot be contained in the pyramidal tract, and it is not likely that the cerebellar tract conducts sensation from the limbs. Hence it is to the anterior part of the lateral column and the posterior column that experiment points. It cannot be an accidental coincidence that it is in these two regions that secondary degeneration proves the existence of fibres which conduct upwards, and extend through the spinal cord to the brain. These are in the posterior median columns, and in what I have termed the "antero-lateral ascending tract" (p. 122). The experiments of Schiff suggest that the lateral column is the path of sensibility to pain, and those of Woroschiloff point to the same conclusion, since he refers to it the conduction of

impulses that have a reflex influence on the vaso-motor nerves, and such an influence is probably only exerted by the impulses that excite a sensation of pain. Schiff concluded that the conduction of tactile sensibility is in the posterior columns, and, moreover, in the posterior column of the same side, there being no decussation in the cord. The latter is certainly erroneous, so far as man is concerned. That the path of sensibility to touch, as well as that of sensibility to pain, crosses the middle line is proved by the effects of unilateral lesions of the cord. To these we may now turn, and ask how far they support the suggestions of experiment.

Unilateral lesions of the spinal cord, which cause loss of sensibility of the skin, do so on the side opposite to the lesion. This is true of all forms of sensibility. They thus prove, beyond doubt, the decussation in the spinal cord of the path from the cutaneous nerves of pain (*i. e.* common sensibility), of touch, and of temperature. An analysis of the cases in which both effect and lesion have been exactly ascertained—which will be considered more fully when these lesions are described—suggests certain other conclusions regarding the position of these paths. They suggest, first, that the paths for sensations of pain and of touch are not near together. Sensibility to pain has been lost in almost all recorded cases, but that to touch in only two thirds. They suggest secondly, that the two paths for tactile sensibility are nearer together than are the two paths for common sensibility. In no case of chiefly unilateral lesion has sensibility to pain been lost on both sides, whereas in two recorded cases sensibility to pain was lost on the side opposite to the lesion, while that to touch was lost on both sides.* Both these conclusions harmonise with the suggestion of experiment, that painful sensations are conducted in the lateral column, those of touch in the posterior column. These conclusions, moreover, are supported by the facts of the only two cases that I have been able to find



FIG. 73.—Section of spinal cord between the second and third cervical nerves, contused by gunshot injury.

which afford direct localising indications. One is a case, recorded by myself,† in which the spinal cord in the upper cervical region was damaged by a small spiculum of bone being driven against it by a bullet, which, entering the mouth, lodged in the body of the third cervical vertebra. The extent of the lesion of the cord is shown in Fig. 73. The chief injury is clearly to the lateral column and grey matter, the posterior column

* It is possible that tactile sensibility may have been lost, in other cases, on the second side, *i. e.* on the side of the lesion, and may have escaped notice in consequence of the exaltation of sensibility, which is commonly present, and which is presumably due to the nerves of common sensibility.

† 'Clinical Society's Trans.,' vol. xi, 1878, p. 24.

being merely swollen, apparently by œdema. The affected part was the seat of hæmorrhage and hæmorrhagic infiltration. The effect was entire loss of sensibility to pain on the opposite side without any impairment of tactile sensibility. To this the other case, recorded by Müller,* is almost a complement. A stab-wound divided the whole of one half of the cord, including the posterior column, and also the posterior column of the other side almost up to the nerve-roots. The whole of both postero-median columns were thus divided. The effect was loss of sensibility to pain on the side opposite to the lesion, and of that to touch on both sides.

From all the evidence, direct and indirect, it seems to be almost certain that the antero-lateral ascending tract constitutes the path for sensibility to pain, and to this conclusion no important facts are opposed. But it is otherwise with the other conclusion, also strongly suggested by the evidence we have reviewed, that the posterior median column contains the path for sensibility to touch. Regarding this column there is another class of facts, suggesting a different function for its fibres, and these facts must receive careful consideration before we can draw any conclusion regarding its relation to tactile sensibility. The column is often found diseased, and intensely diseased in some parts, when there is no loss of any form of cutaneous sensibility. Moreover, certain facts seem to indicate another function for the fibres of the median columns. We have seen that the way in which their ascending degeneration spreads out towards the periphery of the cord, just above a lesion, and reaches the posterior root-zone, suggests that posterior root-fibres pass directly to this column without decussating. There is one form of sensibility the path for which seems not to decussate in the cord—the sensibility of the muscles. The facts, old and recent, of unilateral lesions of the cord (considered more fully in the next section) supply conclusive evidence that the sensibility of the muscles is lost on the side of the lesion, as was first pointed out by Brown-Séquard. These non-decussating fibres of the posterior median columns may constitute the path for this sensibility. In harmony with this view is the fact that these columns are diseased almost constantly in locomotor ataxy (q. v.). But, even if we admit this theory as probable, we are not justified in assuming that this is the sole function of these columns. Fibres pass to them also in other ways, from the posterior commissure in the middle line, and from the neck of the posterior cornu (see Fig. 71), and, as the figure shows, some of these fibres may readily come from the posterior commissure, and both these sets of fibres may have crossed in the middle line. Is it possible that these fibres conduct tactile sensibility, and that they are mingled with those that conduct

* 'Beitrage zur Path. Anat. und. Phys. der Rueckenm.,' Leipzig, 1871. Abstracts, which supplement each other, and, together, give a full account of the case, will be found in Köbner's article on unilateral lesions, 'Dent. Arch. f. klin. Med.,' 1877, Bd. xix, p. 190, and in 'Virchow's Jahresbericht,' 1871, Bd. i, p. 152.

muscular sensibility? This theory, perhaps better than any other, harmonises ascertained facts, but it can only be regarded as a suggestion for future observations. If the path is in the posterior columns, and not in their median portion, it must have a zigzag course, passing in and out of the grey matter scores of times in its upward progress, since no fibres of the external column have a continuous upward course of more than a few centimetres. Such an assumption must be a last resort of theory. The case figured on p. 132 seems to exclude the possibility that the grey matter can contain the tactile path.

The path for sensations of temperature is still unknown. The fact that this sense is frequently impaired with sensibility to pain suggests that the two paths may be near together in the lateral columns. More than this cannot be said.

Flechsig thinks that the direct cerebellar tract conducts sensation from the muscles of the lower part of the trunk. It may be diseased when there is no cutaneous anaesthesia, and is chiefly formed at the spinal level of those muscles. Hence the theory has some probability, although the function of the remarkable posterior vesicular tract, from which the fibres chiefly come, is not known. This view would assign a similar function to the direct cerebellar tract, and to part at least of the posterior median column. The post-pyramidal nucleus, in which the latter fibres end, has also an extensive connection with the cerebellum. It may be that the impressions from the muscle-nerves, conducted by these fibres, influence cerebellar co-ordination, but do not directly affect consciousness as a distinct sensation, although they may indirectly influence our conceptions.

But we have to face another difficulty in our localisation of the sensory path. The same impression that is felt, may excite a reflex action. For this, afferent root-fibres must end in the grey matter of the cord. Are the two functions subserved by the same or by different fibres? A similar question presents itself with regard to the muscle-nerves. If the ascending degeneration in the postero-median column, when the cauda equina is diseased, is admitted, as provisionally it must be, as proof that these root-fibres pass up without interruption, the muscle-reflex action must be subserved by other fibres. For it, however, comparatively few fibres may suffice. With regard to the sensory fibres from the skin, the evidence at present suggests that all undergo interruption in the spinal cord, since the paths have not yet been found degenerated when a lesion has been confined to the nerve-roots. If so, the same fibres probably subserve sensation and reflex action.

We know very little of the serial representation of cutaneous sensibility in the posterior roots. It is very probable, however, that there is a progressive representation of the skin, without relation to the muscles. We know that this obtains in the simpler arrangement of the nerves of the trunk; ascending disease causes an affection of sensation which extends around the trunk at higher and higher levels. In the limbs we have indications of the same relation: the soles and

palms may be affected alone, and anæsthesia may extend on the limb to a certain level, irrespective of nerve distribution. This is often seen in the legs. I have seen anæsthesia in the arms, due to disease of the spinal cord, up to midway between shoulder and elbow, and there cease at the same level around each arm. At the same time, the strictly progressive representation may obtain only within the cord, and be modified in the nerve-roots. Thus, affection of the first dorsal roots causes anæsthesia only in the ulnar region of the palm. Disease of the cord itself may affect sensation on the tips of the fingers only. It is possible that the afferent nerves from the muscles differ from those from the skin, and correspond in origin to the motor nerves to the same muscles.

REFLEX CENTRES.—The grey matter of the spinal cord constitutes a series of reflex centres, some of which must be of considerable vertical extent and considerable complexity, and are subserved probably not only by the grey matter but by the short fibres that in the antero-lateral column (mixed zone and limiting layer) and the postero-external columns (posterior ground-fibres) connect the grey matter at different levels. We have already seen (p. 10) that we must distinguish two forms of reflex action,—the cutaneous reflex action, and the muscle reflex action, the latter producing the “myotatic irritability” which is assumed to determine so-called “tendon-reflex contractions.” The first form of reflex action is not, however, limited to impressions on the cutaneous nerves. It may be produced by stimulation of the nerves supplying the deeper structures, including those of the tendons and muscles. The attempt to obtain the knee-jerk may cause a true reflex action, as well as a local contraction, the two being separated by an appreciable interval of time. In the skin it is apparently subserved by all the sensory nerves. It may be present, although the nerves for either pain or touch are degenerated.

The central process involved in reflex action must take place between the posterior nerve-roots and the anterior roots. The motor ganglion cells must take part in it, and form part of the reflex centre, and the rest of the centre is probably constituted by part of the fibrillary plexus, and perhaps also by sensory nerve-cells. The whole part may be spoken of as the “reflex arc.” Lines of different resistance doubtless exist in the centre, and determine the form of the reflex action. The stronger (within limits) the afferent stimulus, the wider does the process spread in the centre, and the more extensive is the movement produced. Thus a slight touch on the sole may cause only a movement of the foot, while a stronger impression may cause a movement of the whole limb. This is true only “within limits,” because a very strong stimulation may prevent any reflex movement, by inhibiting the centre which a slighter stimulus discharges.

The motor ganglion cells from which the anterior roots proceed,

must form part of the centre for each form of reflex action, but the, afferent impression for the two must be conveyed by different nerves and that portion of the centres which intervenes between the posterior nerve-roots and the motor ganglion cells, may be distinct. Beyond this, we can say nothing of the relation of the two centres. The muscle-reflex action must be the less complex. It is a simple effect of the stimulation of certain fibres on certain nerve-cells, strictly limited to the cells related to the muscle from which the afferent impulse comes. The cutaneous reflex process is far more considerable in degree, wider in range, and more complex in character. In some of the lower animals it may attain the elaborateness of a purposed movement.

These reflex centres are subject to control. That for cutaneous reflex action must be controlled by a cerebral centre, since, when all cerebral influence is cut off, as by a transverse lesion in a higher part of the cord, the action of the lower spinal centres becomes excessive. We have no precise knowledge of the seat of the controlling centre or of the path by which the control is exerted. The mechanism must, however, be very complex, since many cerebral lesions, which cause hemiplegia, cause also a diminution of cutaneous reflex action on the paralysed side. This effect seems to indicate that the cerebral centre which controls reflex action, is itself habitually controlled by a higher centre, perhaps in the cortex. When this is destroyed, or the path from it is interrupted, the controlling centre passes into a condition of increased activity, and the reflex action is lessened. This is a theory only, but it seems impossible otherwise to explain the facts.

The muscle-reflex centres seem also to be under the control of other centres, because, if there is disease of the cord higher up, they also pass into a condition of increased activity. But the phenomena of their disturbance presents three important points of difference from those of the cutaneous reflex action. (1) We know with certainty the path, disease of which determines their excess: it is the pyramidal tracts. (2) Disease of the brain never causes a permanent diminution in the action of the spinal centres, as it so frequently does in the case of the centres for cutaneous reflex action. On the contrary, disease of the pyramidal fibres in the brain causes the same excess of this form of reflex action as does disease of the pyramidal fibres in the cord. (3) The excess does not, as a rule, quickly follow an interruption of the path from the brain, as does the excess of superficial reflex action. It is manifested at the end of a week or ten days, and gradually increases. Two explanations of the lateness of the manifestation of the excess are conceivable. The lesion of the pyramidal fibres causes also their degeneration below the lesion, and this must involve their termination in the grey matter. The excess of action of the centres may be the result of the degeneration of this terminal ramification, which may be the controlling structure that normally restrains the activity of the centres. In this case, the time before the excess is manifested may be that needed for the degenerative changes to

descend the fibres to the level of the centres concerned. Secondly, the mere arrest of the central influences passing down the pyramidal fibres may cause the excess of the muscle-reflex centres, and the interval which elapses before the excess is manifested may be due to the fact that there is little tendency to increased activity, that but slight habitual control is exercised or needed, and that the capacity for over-action is gradually developed and gradually increases. On the whole, it seems to be highly probable that the normal restraint is due, in some way, to the influence of the terminal portion of the pyramidal fibres within the grey matter, and that the excess of this reflex action follows any impairment of the integrity of these structures. This may not be, however, the only cause of excessive reflex action. A primary over-action of the centres is conceivable, and may occur in some conditions, but it is not probable that such over-action is the cause of the permanent excess which results from disease higher up the spinal cord.

The chief local forms of superficial and muscle-reflex action have been already described (see pp. 11, *et seq.*). Their centres are found in the corresponding segments of the cord here enumerated. The superficial reflex centres are, however, extensive, and may be excited through a wider region than that indicated. (See also p. 142.)

Superficial reflex action.—Plantar, second sacral; gluteal, fourth lumbar; cremaster, second lumbar; abdominal, sixth to eleventh dorsal (epigastric, sixth dorsal); scapular, fifth cervical to first dorsal.

Muscle-reflex action.—Calf muscles (foot-clonus), fifth lumbar and first sacral; knee-jerk, third and fourth lumbar; flexor digitorum, triceps, seventh cervical; biceps, supinator longus, sixth cervical.

CO-ORDINATION OF MOVEMENT.—The co-ordination of movement is certainly in part determined by the spinal cord. It is a complex function, probably depending on both motor and sensory arrangements, but chiefly on the impressions from the sensory muscle-nerves. The consideration of its mechanism may conveniently be postponed until the discussion of its defect.

INFLUENCE ON NUTRITION.—The nutrition of the *muscles* is under the control of the anterior grey matter, and probably of the motor nerve-cells. The influence is exerted through the motor nerves, and indeed consists in the normal conducting integrity of the fibres (see p. 21).

The nutrition of the *bones and joints* is also under the control of the cord, but we do not know by what part that control is exerted. The growth of the bones is hindered when the anterior cornu are diseased, but no change seems then to occur in the osseous structure. Such a change, and an alteration in the joints, may occur in locomotor ataxy, in which there is disease of the posterior nerve-roots.

The nutrition of the *skin* seems to depend on nerves that have their course in the posterior roots, but we do not know whether there are special trophic nerves, or the position of the centres by which the influence is exerted. The clearest fact is that irritation of the nerve-structures has far more effect than simple loss of function. It is when the cord or nerve-roots are the seat of irritative inflammation that the most serious trophic changes occur.

VISCERAL CONTROL.—Although the viscera are under the immediate control of the sympathetic system of nerves, they are related to centres in the spinal cord, and the relation is the most direct and important in the case of the organs over which the will has an influence, the *rectum* and the *bladder*. The centres for these are in the lumbar enlargement, but we do not know their exact position. They are probably complex reflex centres. We can best understand their action by studying them when voluntary influence is lost.

The centre for the sphincter ani is the more simple, but the system of action of each may be the same. In the wall of each viscus we have muscular fibres to expel the contents, and at the mouth a sphincter-arrangement to prevent their continuous evacuation. Fæces or air in the rectum, and urine in the bladder, may excite the lumbar centre, and cause two effects—contraction in the wall and relaxation of the sphincter. This process can be controlled by the will to a considerable extent, although we are still ignorant of the precise mode in which the voluntary influence is exerted. But if the volitional path in the cord is interrupted above the lumbar centres, the will can no longer control the reflex processes; as soon as fæces irritate the rectum, they are expelled by the reflex mechanism; as soon as a sufficient quantity of urine accumulates in the bladder, a reflex contraction of the detrusor, and relaxation of the sphincter, cause its escape. The affection of the voluntary path for the sphincters is not always proportioned to that for the legs. If the damage to the cord involves also the sensory tract, the patient is unconscious of the action of the bladder or bowel. If the sensory tract is unaffected, the patient is aware of the process, but cannot control it. It is often said that there is permanent relaxation of the sphincters, but this is true only when the lumbar centres are inactive or destroyed. In this condition, evacuation occurs as soon as fæces or urine enter the bowel or rectum. The urine escapes continuously, instead of being expelled at intervals. The condition is less obvious in the case of the rectum, because there is not such continuous passage of fæces into the rectum as there is of urine into the bladder. We may, however, distinguish between the two states of the rectum by the introduction of the finger. If the lumbar centre is inactive, there is a momentary contraction, due to local stimulation of the sphincter, and then permanent relaxation. If, however, the reflex centre and motor nerves from it are intact, the introduction of the finger is followed, first by relaxation, and then by gentle, firm, tonic

contraction. I have verified this by introducing an india-rubber cylinder, instead of the finger, and registering the pressure on the

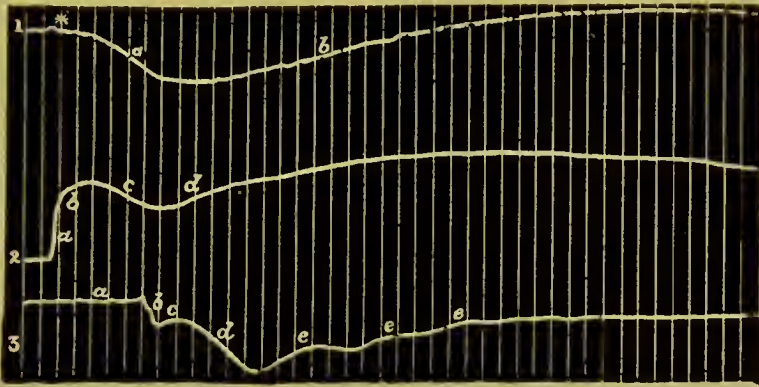


FIG. 74.—TRACINGS OF THE ACTION OF THE SPHINCTER ANI.
The vertical lines represent seconds of time.

1. Effect on contraction of sphincter of the injection into the rectum of a small quantity of air at *. *a*, Fall in pressure due to the inhibition of the contraction; *b*, rise due to the slowly returning contraction. 2. Effect of the introduction of the instrument. *a*, Sudden rise of lever at moment of introduction, due to the exposure of the instrument to the pressure of the sphincter (the top of this line represents the degree of previous contraction); *b*, initial rise due to increased contraction; *c*, fall from partial inhibition; *d*, subsequent contraction, rising to a greater degree than the initial contraction, and subsequently falling slightly. 3. Effect of cough. *a*, Pressure of tonic contraction of sphincter (the slight irregularities are due to pulse-waves); *b*, fall in pressure, due to the movement of the instrument by the cough; *c*, initial contraction; *d*, relaxation of inhibited sphincter; *e, e, e*, rhythmical variations in subsequent rise. (From the 'Proc. Royal Society,' 1877.)

cylinder by connecting it with a recording apparatus. The relaxation is then found to be preceded by a very slight brief contraction, and to be followed by unbroken tonic contraction. The relaxation may also be readily produced by any impression on the mucous membrane above the sphincter. Fig. 74 shows some of the tracings obtained.

The action of the bladder mechanism can be best understood by assuming that the motor centre really consists of two parts, one (*ms*, Fig. 75) maintaining the contraction of the sphincter, the other (*md*) exciting the contraction of the detrusor fibres, and that these two parts

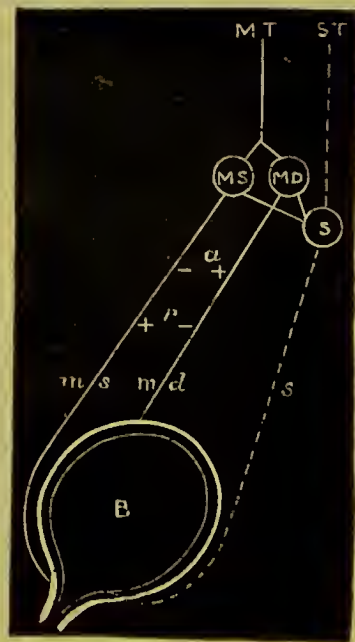


FIG. 75.—DIAGRAM SHOWING PROBABLE PLAN OF THE CENTRE FOR MICTURITION.
MT, Motor tract, *ST*, sensory tract in the spinal cord; *MS*, centre, and *ms*, motor nerve for sphincter; *MD*, centre, and *md*, motor nerve from mucous membrane to *s*, sensory portion of centre; *B*, bladder. At *r*, the condition during rest is indicated, the sphincter centre in action, the detrusor centre not acting. At *a*, the condition during action is indicated, the sphincter centre inhibited, the detrusor centre acting.

are antagonistic: when one acts the other is inhibited. Thus, in normal rest, the sphincter centre is active, the detrusor at rest. Action is produced by an afferent impulse from the bladder and a voluntary impulse from the brain. Then the detrusor centre acts, and the sphincter centre ceases to act. If voluntary power is impaired, the afferent impulse from the bladder may be insufficient, and then there is retention, or in other cases the motor centre may yield too readily to the afferent impulse, and there is reflex incontinence.

Although no other part of the *alimentary canal* is under voluntary influence, all parts are probably related to the spinal cord, by means of the connection between the sympathetic system and the nerve-roots. Constipation is extremely common in disease of the spinal cord, and is often greater than can be accounted for by the mere loss of power of the abdominal muscles. The pain in the back that is so common in disease of the stomach, has been regarded as a reflected pain, due to this connection.

The *uterine* functions are probably independent of the cord in greater degree than those of any other organs under the influence of the sympathetic. The function of menstruation goes on independently of cord disease and may be regular although there is absolute arrest of all conduction, sensory and motor. Pregnancy and labour may proceed in normal course, in spite of such disease of the cord as causes complete motor paraplegia.

Sexual Functions.—The activity of the sexual organs depends on the integrity of the reflex loop to and from a special centre, also situated in the lumbar enlargement, but the due action of this centre depends on cerebral (psychical) as well as on reflex influences. Disease of the centre, or of the nerves leading to or from it, abolishes sexual action. The sexual reflex is, however, one of the cutaneous reflexes, and it shares the condition of these rather than of the muscle-reflex processes.

The centre is probably double, and its action is impaired by interference with either half. When, by disease higher up, the connection with the psychical centres is interrupted, the sexual act cannot be perfectly performed. If the path from the controlling centre (p. 136) is unimpaired, the reflex sexual processes are not in excess, and may even be diminished; but if the path from this controlling centre is also interrupted, the reflex sexual processes are in excess like the other superficial reflexes, and priapism results. If the reflex centre, or connecting paths, are partially diseased, the sexual process may be impaired and imperfect, but not abolished.

Vaso-motor Centres.—The sympathetic nerves to the vessels are influenced from the spinal cord. We have no definite knowledge of the position of the centres or paths from and through which this influence is exerted, but some facts of disease suggest that the centres may be situated in the intermediate grey matter. The action of the vaso-motor nerves may be deranged by disease of the cord. It is pro-

bable that such a relation exists between all parts of the vaso-motor system of nerves and the cord, and that the relation obtains between each segment of the cord and the vascular nerves to the corresponding part of the trunk and limbs. This derangement in disease may take a part in the production of the changes in nutrition, but it is probably not the sole mechanism by which these changes are produced.

There is a special connection between the sympathetic and the cervical region of the spinal cord. This part contains the path to the visceral nerves that control the sugar-forming functions of the liver. The lower part of the cervical enlargement contains also an important centre which controls the sympathetic nerves for the vessels of the head and face. The path of central influence over the radiating fibres of the iris passes also from the region of the third ventricle, down the cervical cord, by the lowest cervical or first dorsal nerve, to the sympathetic, and then ascends the cervical trunk, and passes along the fibres which accompany the internal carotid artery to the ciliary ganglion. Fibres also pass from the cervical cord to the sympathetic through which the action of the heart is accelerated.

MUTUAL RELATION OF THE FUNCTIONS OF THE SPINAL CORD.—It may be well to present, in the form of a table, the relation of the several functions of the cord, motor, sensory, and reflex, as far as our knowledge of these relations extends. Many points are still uncertain; these have been, for the most part, omitted. The table does not need any detailed explanation. It is merely a comparative collection of the facts that have been stated in the preceding pages. We shall find the value of this collocation of functions when we consider the effects of transverse lesions of the cord at different levels.

*SYMPTOMS OF DISEASE OF THE SPINAL CORD: INDICATIONS
OF THE SEAT OF THE LESION: ANATOMICAL DIAGNOSIS.*

The symptoms of disease of the spinal cord consist in derangement of its various functions; the loss of some, the exaltation and perversion of others. We have already considered these functions, and the parts of the cord by which they are subserved. We may now briefly consider the character of their derangement and its significance in regard to the position of the disease. The combination of symptoms indicates the seat of the lesion; we infer its nature from their mode of development, and other considerations, which will be subsequently discussed. It is always important to keep these two elements of the diagnosis distinct in the mind.

A common feature of the symptoms of disease of the spinal cord is their bilateral character. This depends on two causes. First, the structures in each half of the spinal cord are in close proximity, and any random process, such as inflammation or tumour, readily affects both halves. Secondly, morbid processes that commence in the nerve-elements, and affect them according to their function ("system diseases") usually involve the corresponding structures of the two sides. Hence the characteristic type of motor palsy is paraplegia. The vertical extent of the bilateral symptoms depends on the position of the disease. Here, however, we must distinguish two classes of symptoms. Those which depend on the interruption of the conducting path, to or from the brain, involve the whole region below the disease. Those which depend on damage to the centres in the cord, are present only at the level of the lesion.

But disease of the cord does not always cause bilateral symptoms. A random process, of small extent, may damage one side only. It may affect one or several structures on one side. Such a process never affects all parts of one side without damaging, in some degree, the other side, except in very rare forms of traumatic lesion. Again, "system diseases," in quite exceptional cases, are limited to one side, and, much more frequently, they may affect one side earlier and more than the other.

MOTOR SYMPTOMS.—*Loss of motor power* results from interruption of the motor path in any part of its intra-spinal course,—pyramidal tracts, anterior grey matter, anterior nerve-roots. We have seen that the path may be divided into two segments, upper and lower (see p. 116). The spinal cord contains a considerable portion of the nerve-fibres of the upper segment, and their lower termination in the grey matter, but only the commencement of the lower segment, the motor ganglion cells, and root-fibres proceeding from them. The chief part of the lower segment is outside the spine, in the nerve-trunks. So far as

the loss of power is concerned, the effect is the same, in whatever part of the motor path the interruption is situated, but the other symptoms that accompany the loss of power differ very much according as the interruption is in the upper or lower segment. The lower segment influences muscular nutrition, and forms part of the path of reflex action. Hence, as we shall presently see, the muscles waste, and reflex action is lost, when this is diseased; but when the upper segment is damaged, there is no muscular wasting, and reflex action, instead of being lost, is commonly increased. The symptoms produced by disease of either segment are essentially the same, whatever part of the segment is diseased, whether the cell at the upper extremity, the fibre, or the ramification at the lower extremity of the fibre. If, for instance, the intramuscular nerve-ending is paralysed by curara, the symptoms produced are the same as if the fibre in the nerve is divided or the cell in the cord is destroyed. Disease of the termination of the upper segment, in the grey matter of the cord, must, of necessity, produce the same effect as disease of the pyramidal fibre itself, or the cell in the cerebral cortex from which the fibre springs. We shall afterwards see that this consideration has very important applications.

If the fibre of either segment is divided, it degenerates below the division, since its nutrition depends on the influence of the cell from which it has sprung. It is probable that the nutritional stability of the fibre, or rather of its essential element, the axis-cylinder, becomes less, and more easily deranged, the greater the distance from the parent cell, and that it is least in the terminal ramification of each segment. This may be the reason why curara acts chiefly on the intramuscular nerves. If this is true of the upper segment, many facts of pathology become clearer to us.

A lesion of the pyramidal tract causes loss of power in all parts below the level of the disease, that is to say, of all parts the fibres for which are interrupted. A lesion of the grey matter, or of the anterior roots, causes paralysis only of those parts which are, functionally, on the level of the lesion. The two mechanisms often coincide. A transverse lesion in the cervical enlargement, for instance, may cause paralysis of the arms from the damage to the grey matter and nerve-roots, and paralysis of the legs from damage to the pyramidal fibres. The disease involves, in the former case, the commencement of the second segment of the motor path for the arms; in the latter, the middle of the upper segment. The associations of the paralysis differ accordingly. The parts affected by a lesion at any level will be readily ascertained by an examination of the table of functions on p. 142.

• It is important to note that the affection of motor power is often incomplete. It may then involve one set of muscles more than another. The flexors or the extensors may be chiefly paralysed. In disease of the dorsal cord, it is very common for the flexor muscles of the hip and knee to suffer more than the extensors. Why this should be we do not know, but the fact is important, because con-

siderable weakness of the flexors of the knee is readily overlooked. In examining motor power, each set of muscles should be separately tested.

Motor over-action ; Spasm is frequent in disease of the spinal cord and its membranes. In the latter case it seems to be the result of direct irritation of the motor structures. In some acute lesions of the cord itself, spasm seems to have a similar origin. In chronic disease the mechanism is certainly different, and apparently depends on over-action of the reflex centres, due, not to irritation, but to deficient control. The spasm may preponderate in either the flexors or extensors, so that the legs, when rigid, may be drawn up or straight out. The flexor spasm seems to be due to an over-action of the centres for cutaneous reflex action, the extensor spasm chiefly to that of the centres for muscle-reflex action, although it may also be excited by a cutaneous impression. The latter will be better understood when the disturbance of this form of reflex action has been described.

Muscular contractions, shortening of muscles, occur in three different ways. (1) As a result of long-continued irritation of motor fibres, chiefly of those of the nerve-roots. This form has been already described and illustrated in the account of diseases of the nerves (p. 78, Fig. 50). (2) From long-continued spasm, shortening of muscles may occur. Usually the contraction is facilitated by weakness of the opponents or by posture. It is especially common in the flexors of hip and knee, in cases in which flexor spasm predominates, and in the extensors of the ankle when there is extensor spasm. The last is aided by the extended position of the foot in the recumbent posture. (3) Whenever one group of muscles is much more paralysed than their opponents, the latter become contracted, although spasm is absent. Such contraction is common in atrophic spinal paralysis, and gives rise to serious deformities. The active contraction of muscles in all these forms may ultimately result in actual tissue changes in them, so that they cannot be passively extended except by the application of great force. This condition must be carefully distinguished from active contracture.* In the latter, gentle extension, kept up for a few minutes, restores the muscle to its normal length. All forms, and especially the first two, begin as an active contracture, and it is only after this has continued for some time that the tissue changes occur, which prevent extension. In the third form, however, the tissue changes develop much more quickly than in the second.

Inco-ordination of movement, although a motor symptom in character, is probably sensory in origin. It occurs chiefly in disease of the posterior columns and posterior nerve-fibres. This is its chief significance. It has also been met with in rare cases of unilateral lesion of the cord. Its probable mechanism will be presently mentioned, and will be discussed more fully in the account of locomotor ataxy.

* The word "contracture" is applied to persistent shortening of a muscle, depending on active contraction of the fibres, and not on tissue changes.

SENSORY SYMPTOMS.—Loss of sensation is a very common effect of disease of the spinal cord. It may be total, and involve all forms of sensibility, or partial and affect only some forms. The statements made on pp. 6-10 regarding impairment of sensation generally, and the method of ascertaining it, apply to that which results from cord disease, and need not be here repeated. It has been also pointed out (p. 130) that loss of sensation occurs less readily than loss of motor power, so that it is common for complete muscular paralysis to co-exist with intact sensibility. The latter may be impaired by disease of any part of the sensory path,—posterior roots, probably also the posterior cornua and commissure, or the conducting tracts in the cord. A division of the sensory path into upper and lower segments is conceivable, analogous to that of the motor path, although we have not the same clear ground for the distinction. Disease of the posterior nerve-roots causes loss of reflex action as well as loss of sensation, just as disease of the anterior roots interferes with reflex action as well as with motor power. Interruption of the sensory conducting tracts higher up leaves reflex action unaffected. A focal lesion, such as transverse myelitis, may damage both the conducting tracts and the nerve-roots, or only one of these. Disease outside the cord, compressing it, often has the same double effect. Hence it is important to test sensation at the level of the lesion, as well as in the parts below. Areas of anæsthesia may thus be found on the trunk when there is none in the legs. Considerable disease of the nerve-roots outside the cord may impair all forms of sensibility, although slight damage may arrest the conduction of tactile impressions, and not those of pain, which are probably more energetic. Disease of the roots within the cord often causes only partial loss, probably because the fibres which conduct different impressions have a different course. Disease higher up the cord still more frequently causes partial loss; either sensibility to pain or to touch may be impaired. That to temperature is rarely affected without that to pain. We do not yet know precisely the significance of the special form of loss. It is highly probable, however, that loss of sensibility to pain is produced by disease of the antero-lateral ascending tract (p. 131). Future observations must determine whether loss of tactile sensibility can be regarded as evidence of disease of any part of the posterior columns.

It is probable, however, that disease of the posterior median column and cerebellar tract impairs the conduction of impressions from the muscles, although it is doubtful to what extent the impressions thus conducted influence consciousness, and how far their arrest entails a distinct sensory loss. They may pass up to the cerebellum and influence the co-ordinating function of its middle lobe, and even influence indirectly the cerebral processes which subserve conceptions of movement and posture, without giving rise to any conscious sensation. At the same time it is certain that sensations from the muscles may influence consciousness directly, as the pain of cramp sufficiently

shows. The sensory muscle-nerves are apparently influenced by pressure and extension; and impressions from them, determined by the adaptation of the muscles to their elongation or shortening on passive or active movement, are the chief elements in the sense of posture. The methods by which this sense can be tested have been already described (p. 9). Its loss is more common in disease of the nerves or nerve-roots (as in tabes), than in disease of the conducting tracts, but is often very marked in cases of unilateral lesion of the cord (see p. 156) and then exists only on the side of the lesion. The sense of posture seems to be impaired more readily, and by a slighter lesion, than the sensitiveness of the nerves to external pressure or extreme contraction of the fibres, whether that of cramp or produced by electricity. The impairment of the function of these nerves seems capable of causing inco-ordination of movement (see p. 157). Perhaps this is due to the interruption of the fibres that go to the cerebellum:

Increased sensitiveness, hyperæsthesia and hyperalgesia, are also common in disease of the spinal cord, and usually depend on irritation of the conducting fibres in some part of the path, but whether the irritation produces the effect by intensifying the impression as it passes, or by exalting the irritability of the related part of the cerebral centre, we do not know. The former hypothesis is tenable, since the phenomena of stimulation of nerves shows that their axis-cylinders have some power of transforming other forms of energy into nerve force, *i. e.* of evolving nerve force, and therefore of increasing the strength of that which passes along them.

The increased sensation may be felt as actual pain, or as "thrilling" or "shock-like sensations." It is probable that the latter are due to increased sensitiveness of the nerves of tactile sensibility. When there is an extreme increase, pain may be produced by a touch, but it is more probable that the touch stimulates the over-sensitive nerves of common sensibility, than that actual pain is produced through the tactile nerves. Other forms of altered sensibility will be described in the account of the diseases in which they occur.

Pain, referred to the spine, occasionally present in organic disease of the cord, is more frequent in disease originating in the meninges or bones. But the frequency with which spinal pain is present in abdominal, especially gastric, disease, and in neuralgic affections, lessens its diagnostic value when it exists alone. Moreover, in cases of organic disease, pain is far less frequent when the disease begins in the cord, than when it commences in the protecting structures. In meningitis, acute or chronic, spinal pain is frequent, and in organic disease of the bones of the vertebral column it is an almost constant symptom, and is combined with local tenderness. The same combination of local pain and tenderness frequently occurs in cases of neuralgic pain, "rachialgia,"—a condition that is often loosely termed "spinal irritation," especially when it succeeds, as it often does, concussion of the

spine. When pain about the spine occurs in organic disease of the cord, it is more often referred to the neighbourhood of the spine, to the loins or the sacrum, than to the spinal column itself. Such pain is occasionally an early symptom of organic disease, but its origin, and precise significance, are not known.

A still more important group of pains are those which are referred to the parts to which the sensory nerves are distributed, and have hence been termed "excentric" or "radiating pains." They are due to the irritation of the posterior nerve-roots in their passage through the intervertebral foramina, through the membranes, or through the posterior columns of the cord. Other similar pains, usually less acute, may be produced by irritation of the sensory conducting tract higher up the cord. These are often dull pains, closely resembling rheumatism, and frequently mistaken for rheumatism by the patients themselves and their medical attendants. The mistake is the more easily made, because other symptoms suggestive of spinal disease may be inconspicuous, and the rheumatoid pains, in acute cases, may be accompanied by febrile symptoms, and in chronic cases may be influenced by weather, being much more troublesome in damp and cold than in fine and dry weather. In all cases, persistent rheumatic pains in the limbs should suggest the possibility of spinal disease, and watch should be kept for such symptoms as local loss of power, or alterations in reflex action. In other cases, especially in locomotor ataxy, the pains are sharp and darting. The position in which these various radiating pains are felt—legs, trunk, or arms—depends (when the nerve-roots are irritated) upon the seat of the disease—at the lumbar, dorsal, or cervical region of the cord. Occasionally the irritation is felt, not as a sharp pain, but as a painful sense of tightness, as if a band were tied tightly around the limb or trunk—the "girdle-pain," as it is called. When there is transverse damage to the cord, at the lowest part of the healthy region there is a state of irritation of the sensory nerves, and this irritation (referred to the nerve-endings) causes the girdle-pain. When the nerve-roots are irritated by disease of the vertebræ, caries or cancer, the pain is sometimes severe, and, in cancer, is especially increased by movement.

Spontaneous sensations, other than pain, are very common in disease of the spinal cord, and are often of considerable diagnostic importance, but suggestive rather of the presence of a morbid state than of its nature. They may present many varieties of character, but the familiar sensation produced by pressure on a nerve-trunk, when the part is said to be "asleep," is the most common and the most significant. It may occur in functional disturbance of the cord as well as in organic disease. It no doubt depends on over-action of the sensory structures, but it has no special significance as to the locality of disease. It may be excited by contact when it is not spontaneous. The still vaguer feeling called "numbness" is also common, and may occur, in slight degree, in diseases of which all other symptoms are

purely motor. All spontaneous sensations are felt most in the extremities of the limbs, on the palms and soles, fingers and toes.

REFLEX ACTION.—*Loss of reflex action* indicates an interruption of the reflex arc concerned. This interruption may be anywhere between the peripheral endings of the motor and sensory nerves, and thus is not necessarily within the spinal-cord, or even within the spinal canal. The position of the interruption must be determined by the associated symptoms; if it is in the centripetal portion of the reflex arc, there is impairment of sensation, since the interruption will equally arrest conduction to the brain. If it is in the centrifugal portion of the arc, there is a corresponding interruption in the path of the voluntary impulse, and loss of motor power. Moreover, disease of the motor centre or motor nerves causes also degeneration of the nerves, and wasting of the muscles. Disease of the reflex centre, or motor nerves, usually causes loss of each form of reflex action, but partial disease of the centripetal path may impair one and not the other, may abolish the muscle-reflex action (myotatic irritability), and not the superficial reflex action, since the sensory nerves for the two are distinct, coming in the one case from the muscles, in the other from the skin. It is also possible that partial disease of the central structures within the cord may impair one and not the other, but we have at present no evidence of the difference in position of the lesion that would cause the difference in effect. It is important to remember that loss of all reflex action may occur as a transient symptom, immediately after the onset of an acute lesion of the cord, from irritative inhibition, and that loss of cutaneous reflex action may occur as a persistent symptom in cases of brain disease.

Excess of reflex action is also very common in disease of the spinal cord. It implies, of necessity, the integrity of the reflex arc concerned. Each form of reflex action may be increased. In some acute diseases, the increase may be due to an irritation of the centres, but in chronic disease it is apparently the result of a loss of control, and indicates disease between the centre concerned and the brain. We have already considered (p. 136) the probable mechanism, and have seen that the excess of the muscle-reflex action is related to disease of the pyramidal fibres, and especially to the degeneration, or altered functional condition, of the terminal part of these fibres, within the grey matter, adjacent to the centres concerned.* This is the signifi-

* It is interesting to note the analogy between the effects of degeneration of the termination of the two segments of the motor path. The over-action, increased irritability, of the muscle-reflex centres, which results from degeneration of the termination of the upper segment, presents some similarity to the increased irritability of the muscular fibres which results from degeneration of the termination of the lower segment. The analogy must not, however, be over-estimated, since the altered mode of response to electricity of the muscular fibres, and wasting of the muscles, seem to show an abnormal state of nutrition, of which we have no indication in the reflex centres of the cord—unless it be in the slow and moderate

cance of considerable excess. Degeneration of the terminal portions of the fibres is commonly secondary to degeneration of the fibres themselves. It is possible, however, that the termination of the pyramidal fibres may be affected without degeneration of the fibres themselves in the lateral column. (This subject will be further considered in the account of primary spastic paraplegia.) The increase of reflex action is chiefly manifested in the legs, in which reflex action is normally more active and important than in the arms. The increase in the cutaneous reflex action is shown by its occurrence on a slighter stimulation of the skin, and by a greater degree and extent of the muscular contractions produced. In these, the flexors usually predominate, so that the leg is drawn up in a strong movement. As the action of the sexual organs depends, in part, on superficial reflex processes, priapism is common when the reflex excess is great. The increase of the muscle-reflex action is shown by an increase of myotatic irritability, and therefore of the so-called "tendon-reflex contractions,"—the increased knee-jerk, rectus-clonus, foot-clonus, &c. These have been already described (pp. 11, *et seq.*).* The excess of this myotatic irritability is at first moderate in degree, and slowly increases. A high degree of over-action is never suddenly attained, as it may be in the centres for superficial reflex action. It would seem as though, in the centres liberated from control, a sort of functional hypertrophy occurs; their nutrition, and capacity for action, responds to the increased demand upon them.

Reflex Spasm.—We have seen (p. 16) that the muscular state on which myotatic irritability depends, assumed to be due to a muscle-reflex action, is probably identical with physiological *tone*. Whenever there is a considerable increase in the irritability, there is a tendency to tonic extensor spasm, which is probably an excessive and morbid degree of the normal tone. At first there is merely slight stiffness of the legs, especially felt on waking from sleep; afterwards the legs, when extended, become distinctly stiff, although they may be quite supple when flexed. If gradually extended, when near full extension the spasm suddenly comes on and completes the movement, as the blade of a pocket knife moves under the influence of the spring. Hence this has been termed "clasp-knife rigidity." Ultimately the extensor spasm may be so great that, when it occurs, both legs are almost as rigid as an iron bar, and are so firmly connected with the

muscular wasting, mentioned in the next section, as occurring after other *irritating* lesions of the pyramidal fibres—a wasting which indicates a slow change in the nutrition of the ganglion cells.

* In addition to the phenomena there described, Morris Lewis has pointed out that a similar contraction in the muscles of mastication can be obtained by striking the chin when the mouth is open, and that this contraction may be excessive when there is a general increase of these phenomena. Lewis called it the "chin reflex" ('Philadelphia Med. and Surg. Reporter,' May, 1886). It has also been described by Beevor ('Brain,' Jan., 1886). De Watteville (*ib.*) proposes for it the name of "jaw-jerk."

pelvis, that if one is lifted from the bed the other is moved also. Paroxysms may be brought on by any impression on the sensory nerves, a prick on the skin, or an attempt to obtain the clonus. The extreme spasm prevents any clonus being obtained, and it may be necessary to flex the limb before an attempt to produce the clonus is successful. As violent spasm is passing off, however, the tension of the muscles may alone set up a clonus, so that the paroxysm of tonic spasm passes into clonic spasm, a phenomenon that was termed by Brown-Séquard "spinal epilepsy," on account of the superficial resemblance to the sequence of spasm in an epileptic fit. This clonic spasm may often be arrested by any strong painful impression on the sensory nerves, a pinch of the skin, or painful flexion of the big toe. There is always much weakness of the legs, when there is much spasm, and there may be complete paralysis. Hence the condition has been termed "spastic paraplegia." It is probable that the central mechanisms concerned in the production of this spasm are those concerned in the reflex act of standing. The spasm often enables a patient to stand, when his voluntary power is quite insufficient for the act. The extensor spasm may be varied by attacks of flexor spasm. The latter are especially apt to come on during sleep.

VASO-MOTOR AND TROPHIC DISTURBANCE.—*Changes in Nutrition.*—Considerable wasting of the *muscles* indicates disease of the motor nerve-cells or of the nerve-fibres proceeding from these cells. If the disease is acute, there is rapid degeneration of the nerves, with the reaction of degeneration—loss of faradaic irritability of the nerves, both within the muscles and outside them, with preservation of the voltaic irritability of the muscular fibres themselves (see p. 45). The wasting in these cases is always great, and there is always loss of reflex action. Occasionally some fibres of the nerve and muscle suffer and others do not, causing the middle or mixed form of reaction (see p. 21). In other cases of disease of the spinal cord, however, there is a slighter degree of wasting of the muscles; there is no loss of irritability in the nerves; the irritability may be slightly increased or lessened, but is changed in the same way to each form of electricity. In this condition there is no loss of reflex action. The change is apparently the result of a peculiar alteration in the nutrition of the nerve-cells. It is usually the effect of an *irritative* degeneration of the pyramidal fibres.

The changes in the nutrition of the skin, if slight and chronic, resemble those produced by disease of the nerves (see p. 52), and suggest disease of the posterior roots. Sometimes, however, these changes are more acute than is ever seen in disease of the nerves. Very slight pressure, continued for a few hours, causes vesication, and even sloughing of the skin. Sometimes bullæ form where there has been no pressure. Occasionally, there is inflammation of the joints. Such intense changes are only met with when the morbid

process in the cord is irritative in its character. The points on which sloughing occurs with greatest readiness are the heel, over the malleoli, the trochanters, and the sacrum. The sacral position of the bedsores in paraplegia constitutes a difference from those which occur in hemiplegia, which form over the gluteal region on the paralysed side.

Vaso-motor disturbance is common in slight degree, irrespective of the seat of the disease, but, like the trophic changes, which probably depend, in some degree, upon it, it is always most intense in irritative lesions of the cord. The slighter and common degree consists merely in increased warmth of the limb in the early stages, with increased redness, while, at a later period, the limbs are colder, paler, and often livid at the extremities. In rare cases intense flushing of the skin has been observed. This has been seen chiefly in cases of disease of the intermediate grey matter. In the same class of cases copious and long-continued perspiration sometimes occurs. When the disease is in the lower cervical region, the flushing and perspiration involve the face and head.

Disease of the cervical cord or nerves is sometimes attended by a change in the pupil, usually one-sided. There may be contraction, from paralysis of the radiating fibres (the sympathetic supply of which is ultimately derived from the cord), or there may be persistent dilatation from irritation of the fibres. The double myosis, so common in some degenerative diseases of the cord, is probably not due to the latter, but is the result of simultaneous degeneration of part of the nucleus of the third nerve.

The frequency of the heart's action is often permanently increased in locomotor ataxy, but we do not know by what mechanism. In disease of the cervical cord (perhaps also in that of the cervical nerve-roots) the heart may be slowed to forty, thirty, or twenty beats per minute, usually only for a time. The effect may perhaps be due to a loss of the influence of the nerves which, when stimulated, quicken the heart, or it may be produced by an indirect upward influence on the centre for the vagus.

VISCERAL AND OTHER DISTURBANCES.—Various disturbances in the *alimentary canal* occur in disease of the spinal cord, and are no doubt due to the fact that the sympathetic nerves immediately controlling the canal are under the influence of the spinal cord, and may be excited to abnormal action by its irritation, or may underact when its influence is lessened. Thus difficulty of deglutition is observed in some cases of acute disease of the upper cervical cord. Vomiting occurs also in disease of the same part, especially in fracture of the cervical vertebræ, and, in paroxysmal form, attends some cases of chronic disease. Constipation is extremely common in disease of the cord above the lumbar enlargement.

Paralysis of the Sphincters.—Incontinence of urine and fæces may

be due to disease of the centres that control the action of the sphincter ani and the muscular mechanism of the bladder respectively, or it may be due merely to disease of the path (probably the pyramidal tract) through which voluntary control is exerted. In the latter case the sphincter ani acts in an automatic reflex manner as already explained. If the finger is introduced within it, the initial relaxation is felt to be succeeded by a firm tonic contraction. This proves that the anal centre in the lumbar enlargement, and the nerves proceeding from it, are intact. If this centre is destroyed, or the nerves interrupted, no tonic contraction can be felt; there is complete and persistent relaxation. The sphincter of the bladder is not accessible to such direct examination, and the mechanism seems to be more complex, but we may distinguish conditions corresponding to those of the rectum. If the lumbar centre is destroyed the sphincter is permanently relaxed; urine dribbles out of the bladder as fast as it enters it; there is simple incontinence. If there is an interruption of the voluntary path, the sphincter may act automatically; when a certain amount of urine has collected in the bladder it excites the muscular fibres in the wall to contract; the sphincter relaxes, and the urine escapes; there is intermittent incontinence. In this condition the mechanism is excited to action by any pressure on the bladder; a movement, or the act of coughing, will make the urine come away. But the muscle of the wall of the bladder is indirectly under the influence of the will; when the sphincter is voluntarily relaxed the detrusor fibres in the wall contract. Loss of voluntary power seems to lead to weakness in the wall of the bladder, so that the bladder is never perfectly emptied, and ultimately the detrusor cannot contract to expel the urine, even when the sphincter is relaxed. Hence retention of urine occurs. When a certain degree of distension of the bladder is attained, the pressure becomes sufficient to force the urine out, whenever the sphincter relaxes, and such relaxation is continually occurring under the high pressure that is continually renewed in consequence of the flow from the kidneys. Hence incontinence results—"overflow incontinence." It is important, if the urine comes away involuntarily, to ascertain which form of incontinence exists; since overflow incontinence, and the retention it indicates, have much graver consequences than simple incontinence. The permanent distension of the bladder constitutes a permanent hindrance to the flow of urine from the kidneys, and serious kidney disease may result. This is a not uncommon cause of death in spinal disease. Moreover, whenever the bladder is habitually emptied imperfectly, decomposition is apt to occur in the residual urine. The decomposition is often aided by the lessened acidity, or even actual alkalinity, of the urine. This change in the composition of the urine excites inflammation of the bladder; pus is formed by the mucous membrane, and this, in its turn, increases the decomposition of the urine. The damage to the kidneys, by the hindrance to

the flow of urine from them, may be increased by inflammation ascending the ureters from the bladder.

Pyrexia.—The general body heat is sometimes changed in disease of the cord apart from the influence of the kind of lesion. Disease of the upper cervical cord, like that of the medulla, sometimes causes hyperpyrexia, and this may also result from acute central lesions that occupy a considerable vertical extent, such as a central hæmorrhage.

Convulsions.—Lastly, convulsive attacks have been known to attend the onset of acute lesions in various parts of the cord, in adults as well as in children. They are most frequent in disease of the cervical cord, but I have known a convulsion to occur at the onset of myelitis in the lower dorsal region. The convulsions are general. Their mechanism is unknown, but in connection with them the readiness with which hemisection of the cord renders a guinea-pig epileptic may be borne in mind.

COMBINATION OF SYMPTOMS.—Certain symptoms are frequently combined, and their combination has a definite localising significance. Loss of motor power occurs in two different associations, according to the position of its cause in the motor path. It may be combined with muscular wasting, loss of electric irritability, and loss of all reflex action. In this case the disease is in the anterior cornua or nerve-roots, the lower segment of the motor path. On the other hand, there may be no wasting, no change in irritability, and, instead of a loss of reflex action, the myotatic irritability is increased. The interruption of the motor path is then in the upper segment, usually in the pyramidal tracts. It may be a focal lesion of the cord, or a primary degeneration of the tracts. The distinction is that, in the latter case, there is nothing more than the muscular weakness and increased myotatic irritability. In the former case there are, or have been, indications that the lesion has extended beyond the purely motor elements.

The symptoms caused by disease of the several elements of the cord have now been described, but it is necessary to consider further the combination of symptoms that results from a total transverse lesion of the cord, and from a unilateral lesion.

A *total transverse lesion*, however limited in vertical extent, separates from the brain all parts below the lesion, and hence, so far as will and perception are concerned, it produces the same effect as if the whole of the cord below the lesion were destroyed. A section across the cord in the middle of the cervical enlargement, for instance, abolishes motion and sensation in all parts below the distribution of the cervical plexus and the phrenic nerve. Hence the extent of the motor and sensory paralysis indicates only the upward limit of the lesion. This is also indicated by the position of the girdle-pain, and radiating pains, or zone of hyperæsthesia, which are due to the

irritation of the sensory nerves in the lowest part of the upper segment. It is desirable to know the symptoms that occur when a transverse lesion is at different heights in the spinal cord. They may readily be ascertained by an examination of the table given at p. 142. There is, however, some uncertainty as to the effect on sensation in the limbs, of disease at different parts of the lumbar and cervical enlargements, since, as we have seen, the exact representation of sensation in the cord is still uncertain.

The upper limit of the lesion is shown by the upward extent of the motor and sensory loss, according to the first three columns. The lowest nerves supply the anus and perineum. Those that supply the skin and muscles of the leg and foot arise from the fourth lumbar to the third sacral segments, and are damaged by a lesion involving the lower part of the lumbar enlargement. We must, however, remember that the skin of the inner part of the leg is not supplied from the sacral nerves, and so may escape when the outer part of the leg, and back of the thigh, have lost sensation. In the middle of the lumbar enlargement the nerves arise which enter the lumbo-sacral cord, and these are probably destined for the flexors of the knee, and for the hip-muscles supplied by the sacral plexus, the glutei, quadratus, and gemelli, and for the skin of the lower part of the gluteal region. These parts then will be paralysed by disease in the middle of the lumbar enlargement, while the muscles and skin in front of the thigh are unaffected. The latter suffer when the disease affects the upper part of the lumbar enlargement, the origin of the anterior crural (rectus, &c.), and obturator (adductors). The skin on the upper and outer parts of the thigh loses sensibility, with the part adjacent to the scrotum, and in the groin, only when the disease damages the highest part of the lumbar enlargement, from which the second and third lumbar nerves arise, and then the flexors of the hip become paralysed. In proportion as the disease is higher in the dorsal region, we have the symptoms ascending higher up the trunk, and marking accurately the height of the lesion by the loss of cutaneous sensibility, and by the impairment—first, of the abdominal muscles, and then of the intercostal muscles. The umbilicus corresponds to the tenth dorsal nerves, and the ensiform area to the sixth and seventh. When the disease reaches the lowest part of the cervical enlargement (the first dorsal nerves), we have the first symptoms in the upper extremity; but these are not, as might be expected, in the muscles moving the shoulder-joint, but in the hand. The first numbness is complained of in the little finger, and the first weakness is in the intrinsic muscles of the hand. Ascending higher, the symptoms pass up the arm with some uniformity, and without respect to nerve distribution. When the middle of the cervical enlargement is reached (the fifth, sixth, seventh cervical) the shoulder-muscles and the serratus magnus become paralysed, and there is general loss of power and sensation. (For details see table, p. 142, and also p. 128.)

Above the level of the sixth pair, the trapezius and sterno-mastoid become somewhat weakened, for the fibres of the spinal accessory which supply them undoubtedly arise in part from this region of the cord. At the fourth and fifth cervical the lower part of the neck becomes anæsthetic, and the diaphragm ceases to act. Here our localisation might cease, for total transverse lesions at this spot necessarily cause death. For a little time the sterno-mastoids and scalmi can still get some air into the chest, rarely in sufficient amount to maintain life for more than a few days. But limited lesions may occur higher up, and then we have complete powerlessness of the muscles moving the head, upper part of trapezius and sterno-mastoid, and other muscles attached to the occipital bone, and interference with sensation in the neck and parts of the head, which are not supplied by the fifth nerve.

The extent downwards of the lesion, its vertical extent, is thus not indicated by the impairment of the conducting functions, by the motor or sensory paralysis; to ascertain it we have to examine the functions of the cord as a central organ, and to ascertain how far they are impaired in the paralysed region--to examine especially muscular nutrition and reflex action. The state of muscular nutrition and irritability indicates how far the anterior cornua are injured. The relation of the several groups of muscles to the cord is shown in the first column of the table. The integrity of reflex action indicates the integrity of the reflex loops, and the study of the superficial reflexes of the trunk is especially instructive in this respect. The series of reflexes, and the relation of each to the cord, are shown in the third column of the table; the myotatic contractions are printed in italics in the position which corresponds to the centres and nerve-roots which are essential for their production. Excess of superficial reflex action indicates withdrawal of the cerebral controlling influence of the reflex centres, and marked excess of the muscle-reflexes suggests the existence of a descending degeneration in the lateral columns, since it implies impaired function of the lowest part of the pyramidal tracts.

Unilateral lesions interrupt the motor path to the muscles on the same side as the lesion, causing one-sided palsy, termed "spinal hemiplegia," when the disease is so high as to affect both arm and leg, "hemi-paraplegia" when it is lower, and affects one leg only. There is often some loss of power on the opposite side, which may be due either to slighter damage to the other side of the cord (since few lesions are strictly unilateral) or to damage to non-decussating pyramidal fibres. Conversely, the paralysis of the leg may be incomplete when that of the arm is complete, owing to the escape of the fibres for the leg which cross lower down the cord. Sensation is affected on the opposite side, but not quite up to the level of the lesion, because the decussation of the sensory tract is not immediate, but occurs somewhat above the entrance of the nerves. The upper level may vary for different forms of sensibility, in consequence probably of the level of

crossing (in relation to entrance) being different for the several paths. A lesion in one side of the lumbar enlargement often affects sensation on the same side as motion, because it damages the sensory path before it has crossed. In all cases of crossed motor and sensory paralysis, the sensibility of the muscles differs from the other forms of sensibility, and if it is affected on one side, this is the side of the motor palsy and not of the cutaneous anæsthesia.*

The crossed affection of cutaneous sensibility may involve all forms of sensation, or only some of them. Sensibility to pain is almost invariably impaired. The temperature sense is usually affected with that for pain; in only two of twenty recorded cases (in which the affection of sensibility was carefully noted) was the sense of temperature normal, and that of pain impaired, and in neither of these cases was the sensibility to pain actually lost. On the other hand, in one third of the cases tactile sensibility was unaffected and in about one-tenth of the cases it has been impaired on both sides.

Cutaneous sensibility is sometimes impaired in a zone at the level of the lesion, and on the same side, in consequence of the damage to the nerve-roots entering the cord, and above this there may be a narrow band of hyperæsthesia from irritation of the roots at the upper part of the lesion. On the side of the lesion, below the anæsthetic zone, there is, in most cases, a remarkable hyperæsthesia, corresponding in distribution with the anæsthesia on the opposite side. Painful impressions are felt most acutely, and even a touch on the skin, or warm or cold bodies, produce pain. The cause of this hyperæsthesia is obscure. Both it and the opposite loss of sensibility may last for twenty years, and it cannot be ascribed therefore to any irritation by the morbid process. It is perhaps due to an altered action of the cerebral centres, on the opposite side of the brain. There is much evidence of an intimate connection between the sensory centres on the two sides, and it is conceivable that the altered functional state of the centre on the side of the lesion, to which impressions cease to come, may induce in the opposite hemisphere a condition expressed as hyperæsthesia. The condition may be thought of in relation to the peculiar phenomena of transfer in hysterical hemianæsthesia.

Reflex action is increased in all its forms on the side of the lesion,

* This was first pointed out by Brown-Séquard in his important study of these cases. Seepticism has been expressed as to the fact, based on a denial that the test commonly employed, recognition of posture, is significant. Experiments on animals, even monkeys, are inconclusive, but the evidence from cases in man is very strong, not only of the occurrence of the loss, but of the significance of the test. In some cases, moreover, the muscles were insensitive to pressure. An instance recently reported is the following:—A stab in the dorsal region caused loss of cutaneous sensibility in the right leg and motor palsy in the left. On this left side, on which cutaneous sensibility was normal, the sense of posture was absolutely lost, while it was present on the right side on which cutaneous sensibility was impaired. The patient often thought the left leg was flexed when it was extended. Ultimately this leg presented distinct ataxy. (Gilbert, 'Arch. de Neurologie,' 1882, p. 275.)

but the increase only occurs after some days. At first it is lessened or even abolished, no doubt from the inhibitory influence of the irritation of the morbid process.

The side below the lesion is at first, for some days or even weeks, warmer than the other, the difference being usually about a degree Fahrenheit. In the course of time this difference passes away and the side may even be colder than the other.

These symptoms may be shown in the form of a table :

Zone of cut. hyperæsthesia. anæsthesia.	LESION.	
Motor palsy. Hyperæsthesia of skin. Muscular sense impaired. Reflex action first lessened then increased. Temperature raised.	}	Muscular power normal. Loss of sensibility of skin. Muscular sense normal. Reflex action normal. Temperature same as that above lesion.

The vertical distribution of the symptoms varies according to the vertical position of the lesion. Their relations are best marked when the lesion is in the dorsal region of the spinal cord. When it is in the cervical or lumbar enlargement the upper limit is less distinct because of the complex representation of skin and muscle in the nerve-roots.

GENERAL PATHOLOGY AND ETIOLOGY: INDICATIONS OF THE NATURE OF THE LESION; PATHOLOGICAL DIAGNOSIS.

The kinds of disease to which the spinal cord is liable are not specifically numerous, but those which are common present several varieties. The processes are congestion and anæmia, hæmorrhage, inflammation, degeneration, and growths. Hæmorrhage and growths are, on the whole, rare. Congestion and anæmia play an uncertain part in the production of symptoms; their influence has been certainly overrated by some writers, and perhaps under-estimated by others. The most frequent lesions are inflammation and degeneration. These two processes, distinct in their typical forms, blend in their intermediate varieties. Inflammation varies much in character and course. Its effects are, in some cases, produced with great rapidity, in other cases with extreme slowness. It probably commences in most instances outside the nerve-elements, which are secondarily involved. Perhaps some acute processes in the nerve-elements themselves are most accurately regarded as a parenchymatous inflammation. Degenera-

tion consists of two processes, a wasting of the nerve-structures, cells and fibres, and an overgrowth of the connective-tissue elements. The latter leads to the condition termed sclerosis. The name has been given to it, probably, because the process is one that, in other organs, increases their consistence. In the cord it has not often this effect. The newly-formed connective tissue is rarely of greater firmness, and is often less firm, than the nerve-elements it replaces. In degeneration we must distinguish two types. In one the process is limited to structures that have the same function; the degeneration probably begins in the nerve-elements, and the overgrowth of connective tissue is consecutive. The so-called "secondary degenerations" are of this character; but similar changes are often primary. In the other type the degenerative changes are not distributed according to function. They are apparently random in incidence, and probably begin in the connective tissue outside the nerve-elements, which suffer secondarily. Insular sclerosis is an example of this type. It is this form which presents the chief gradation to chronic inflammation.

Softening of the spinal cord is very common. Whenever the nerve-fibres are broken up into disconnected globules of myelin, separated by serum in a sort of emulsion, the consistence of the part is necessarily lessened. Inflammation always causes such a breaking up of the nerve-fibres, and hence inflammation always causes softening as its first effect. Ultimately, connective-tissue elements are formed, which increase the consistence, it may be up to, and even beyond, the normal degree. In a very chronic inflammation the formation of new tissue may proceed *pari passu* with the destructive process, and there may be, at no time, much diminution of consistence. It is in these cases that it is difficult to draw the line between inflammation and degeneration. Does such softening of the cord occur apart from inflammation? The question is not easy to answer. In the brain, necrotic softening is very common as a consequence of arterial obstruction—is indeed the common form of softening. But in the spinal cord we have no evidence of the occurrence of necrotic softening. If embolism occurs, it is excessively rare. Arterial thrombosis, due to atheroma of the walls of the vessels, such as is so common in the brain, probably does not occur in the cord. The arteries are smaller than those in which atheroma is met with in the brain, and the lowness of the blood pressure within them involves the absence of the chief cause of atheroma. It is possible that spontaneous thrombosis may sometimes set up the changes that are now regarded as those of primary inflammation, but we have no direct evidence of this. The question will be further considered in the account of inflammation.

Besides the morbid processes to which the cord itself is liable, it suffers also in consequence of disease outside it. It may be compressed by growths, and by products of inflammation, within the spinal canal. Compression not only causes acute degeneration of the nerve-elements, but usually excites actual inflammation. This "compression-myelitis"

may attain a degree and an acuteness out of all proportion to the causal compression. The membranes may be the seat of hæmorrhage which compresses the cord, or inflammation which irritates it, even when the organ is not invaded in appreciable degree.

The series of recognised lesions of the spinal cord, obvious or microscopic, does not by any means exhaust the list of its morbid states. Changes may occur in the nutrition of its elements interfering with their function, which are, and are likely to remain, altogether beyond our means of detection. Such morbid states are often called "functional diseases," but when prolonged they are probably more accurately conceived as nutritional diseases (see p. 1). Derangements of function that we can regard as purely such are few and rare. It is thus convenient to think of the pathological conditions of the cord under the four types which we considered (p. 2) as presented by diseases of the nervous system generally. The types are not always sharply separated, but are not therefore less useful. (1) *Organic disease*, conspicuous to naked-eye examination, such as a hæmorrhage or tumour. (2) *Structural disease*, presenting only change in tint or consistence to the naked eye, but distinct tissue-changes on microscopical examination. (3) *Nutritional disease*, causing no change recognisable even by the microscope. (4) *Functional disease*, purely such.

In connection with the general pathology of diseases of the spinal cord, it is important to note certain general facts of their causation:— (1) An inherited tendency to disease of the nerve-elements, manifested by such affections as epilepsy and insanity. This cause is chiefly influential in producing the structural and nutritional diseases, beginning in the nerve-elements, and especially, among structural diseases, the "system-degenerations" as they are called. (2) Injury.—A severe concussion of the cord may cause (a) instant grave damage, usually hæmorrhage. Or (b) it may cause no immediate effect, but symptoms may come on at the end of a few days and progress slowly or rapidly. Such symptoms are usually due to inflammation, simple, or secondary to minute spots of injury. Lastly (c), the concussion sometimes seems to pervert the process of nutrition in the nerve-elements. Slow symptoms of impaired function may result, and these may progress until, after months or years, there is actual structural disease. (3) Exposure to cold.—Wet cold is especially effective. The most frequent effect of a severe exposure is acute inflammation, but habitual exposure may produce chronic inflammation, or degenerative disease. The exposure that is effective may be general, or chiefly of the feet and legs, occasionally of the back. (4) Syphilis.—The demonstrated ways in which syphilis interferes with the spinal cord are (a) by a syphilitic growth, compressing or invading the cord, and (b) by chronic syphilitic meningitis, damaging the cord and the nerve-roots. But (c) acute and chronic inflammations of the cord are often met with in syphilitic subjects, and have been thought to be, in many instances, of syphilitic origin. It is doubtful whether they present any syphilitic characters,

i. e. any histological features by which they differ from non-syphilitic inflammations. The evidence of dependence on syphilis is stronger in the case of chronic and subacute disseminated inflammation than in the case of acute myelitis. Lastly (*d*), certain degenerative diseases of the cord are very commonly preceded by syphilis; one of them, locomotor ataxy, so frequently that a causal relation between the two can scarcely be doubted. But these degenerative diseases are certainly not syphilitic in nature; they differ in no respect from the similar morbid processes that occur in individuals who have not had syphilis.

The outline just given of the general pathology and etiology of diseases of the spinal cord will enable us to consider the last element in diagnosis, the nature of the lesion. The seat of the disease is indicated by the combination of symptoms; its nature can only be determined by considering, separately and together, several other points:—(1) The way in which the symptoms came on. (2) The causes that can be traced, taken in conjunction with the known effects of those causes. (3) The seat of the disease, taken in conjunction with the known liability of certain structures to certain lesions. The most important of these elements is the mode of onset, and the other indications should only be used in strict subordination to this.

The time occupied by the onset of the disease is thus the first element in the pathological diagnosis. By “time of onset” is meant the period that elapses between the actual commencement of the symptoms and their attainment of a considerable degree of intensity. We may divide the chief modes of onset into five classes, and classify the most common diseases in relation to them, in the following table:

DISEASE.	ONSET.	DISEASE.
	<i>Sudden</i> (few minutes)	} Vascular lesions.
	<i>Acute</i> (few hours or days)	
Pressure and growths	<i>Subacute</i> (one to six weeks)	} Inflammation.
	<i>Subchronic</i> (six weeks to six months)	
	<i>Chronic</i> (more than six months)	} Degeneration.

A lesion of sudden occurrence, the symptoms developing in the course of a few minutes, is almost always vascular, commonly hæmorrhage, sometimes perhaps vascular obstruction. But a vascular lesion may occupy a somewhat longer time in development—a few hours or days. In acute and subacute inflammation the symptoms come on in the course of a few hours, a few days, or a few weeks. Subacute and chronic inflammation occupies from a few weeks to a few months. Degeneration, in which there is no adequate evidence of any inflammatory process, occupies many months, or it may be years. The symptoms produced by tumours which invade or compress, and by simple

pressure (traumatic causes excluded) are never sudden or very acute, and rarely very chronic, the time occupied by the development of the symptoms varying, according to the nature of the cause, from a fortnight to six months.

It is necessary to consider, however, not merely the whole time occupied by the development of the disease, but also the uniformity of its course. Two or more morbid processes may concur. An initial myelitis, for instance, may lead to a secondary degeneration; and, on the other hand, in degenerated tissues, sudden vascular lesions occasionally occur. Pressure often produces local myelitis, which may be acute or subacute in its development. Cancer of the vertebræ, for instance, often causes rapid myelitis. The whole course of the disease must be ascertained before an inference is drawn, and the possibility of a double process must always be kept in view.

The onset and course of the symptoms thus sometimes enable us to decide at once that a lesion is of a given character, as that one which occurs instantly is vascular, or that one which takes years for its development is degenerative. More frequently they enable us to exclude certain morbid processes, and to restrict the possible lesion to two or three forms. For instance, a lesion which comes on in the course of a few hours must be either vascular or inflammatory. Between these we have to decide by attention to other indications.

In actual diagnosis it is convenient to consider next the indication afforded by the position and distribution of the disease. We consider what diseases occur in this situation, and then which of them have the mode of onset that has been ascertained. This indication involves a knowledge of the various diseases and their seat. The most important consideration is that a wide range of symptoms of uniform character indicates the affection of a definite system of structure, and in most instances a disease commencing in the nerve-elements, and if the onset be chronic we may feel sure that it is a degeneration. On the other hand, the involvement of many functions suggests a random process, such as inflammation or pressure. But this indication is always to be subordinated to the mode of onset. Thus the limitation to a single structure does not exclude inflammation: this may affect, for instance, the anterior grey matter only, and cause corresponding symptoms.

The symptoms may indicate a morbid process limited to one half of the cord, but this does not materially modify the diagnostic method. Almost any process may, in rare cases, be thus limited. System degenerations and acute inflammations are least frequently unilateral, and they never reach a considerable degree of intensity on one side without some affection of the other side. On the other hand, tumours and foci of chronic myelitis are often one-sided, and still more often affect one half of the cord first and then the other.

Indication of disease outside the cord, irritation of certain nerve roots, causing severe local pain, often precedes the symptoms of com-

pression and is an important aid to diagnosis. It shows the existence of a morbid process outside the cord before the cord is involved. But we cannot use even this indication except in dependence on the mode of onset. A disease, as a growth outside the cord, may, as we have seen, not only compress the cord, and cause slow loss of power; it may excite inflammation and cause rapid palsy.

The last element in the pathological diagnosis is the detection of any influence which can be regarded as the cause of the disease in the spinal cord, or any associated condition which may indicate an active morbid process. We have seen that the mode of onset may help us to limit the disease to certain possible forms of lesion; the distribution of the affection may render it probable that it is one or other of these forms; and the detection of a cause and the knowledge of the lesions that cause produces may help us to carry the diagnosis still further. The most important general causes of disease of the cord, and the processes to which they chiefly give rise, have been already mentioned. The causal element in diagnosis is chiefly an application of those facts.

It would be easy to formulate more detailed diagnostic indications. Those given will suffice to indicate the general principles of diagnosis; to do more than this would be to hinder, rather than to help. The only sure ground for diagnosis is a thorough knowledge of the various diseases and their symptoms; without this, elaborate formulæ are useless; with this, they are unnecessary.

The distinction of functional and nutritional disease from organic lesions may conveniently be postponed until the symptoms of the former are specially described.

SPECIAL DISEASES OF THE SPINAL CORD.

DISEASES OF THE VERTEBRAL COLUMN.

Diseases of the bones of the spine fall for the most part within the province of surgery. But there are few of these diseases that do not, among their most frequent effects, interfere with the functions of the spinal cord. Hence an account of the diseases of the cord would be incomplete without some mention of the morbid states that begin in its bony case.

INJURIES OF THE SPINE.

Injuries to the spinal column may consist of punctured wounds (which need not be considered here), concussion (the effects of which on the cord will be considered later), and fracture or dislocation of the

vertebræ. A brief mention of the two last conditions may be given, the reader being referred, for fuller information, to surgical treatises.

DISLOCATION.—Simple dislocation occurs in the cervical region, most frequently at the fifth and sixth vertebræ. It may take place gradually or suddenly: gradual displacement is always secondary to disease of the bones; sudden displacement may occur in disease or from injury. The damage to the cord is always greatest in traumatic displacement of normal bones, because the force needed to produce the dislocation is much greater, the displacement is more considerable, and the obliteration of the canal is more complete. Displacement may occur in any direction: it usually involves both vertebral articulations, but, in rare cases, it is oblique, involving one articulation only. The common causes are violent blows or falls on the head, rarely sudden rotation of the head when a weight is carried upon it. The symptoms are a lateral or forward or backward displacement of the head, so that the chin is in contact with the shoulder or the chest, or the occiput with the nape of the neck. The irregularity of the vertebral spines is usually readily detected. The cord is damaged in most cases, and the symptoms are those of a total transverse lesion in the situation of the luxation. It may be merely compressed, especially in cases of disease, in which the displacement has occurred with little force. The symptoms of paralysis have been known to pass away, in such a case, on the reduction of the dislocation. More commonly the cord is also bruised, with extravasation of blood, and secondary myelitis occurs later. In such cases, if the patients live, there may be anæsthesia or hyperæsthesia below the lesion, with total paralysis of the limbs, and excess of reflex action. In rare instances the cord has been completely divided. In still rarer instances of slight displacement it has not been injured.

Rupture of the transverse ligament, which retains the odontoid process, may permit the latter to compress the cord, and thus to cause instant death. This often results from sudden suspension by the head, as in criminal executions. One of the curiosities of surgical literature is a case related by Petit in which a man, playing with a neighbour's child, lifted it up by the head, and caused instant death by rupturing the transverse ligament. The father of the child, entering at the moment, stabbed the man with a knife, the blade of which passed in between the first and second cervical vertebræ, divided the spinal cord, and the man also fell dead. Rupture of the ligament has also resulted from raising a heavy weight with the head. The treatment of dislocation is too purely surgical to be described here.

FRACTURE.—All organic diseases of the bones, weakening them, predispose to fracture. Apart from disease, the accident is most common in adults, the greater elasticity of the vertebral column in children giving to them a comparative immunity. Its great cause is

a blow or fall on the spine, or sudden forcible flexion. In extremely rare cases a fracture, usually slight, has resulted from a severe muscular exertion. This is an important fact, showing that muscular exertion may injure the spine. Symptoms of such injury, in slight degree, are not uncommon.

Fracture may occur at any part of the spine, but is most frequent at the fifth or sixth cervical, and at the last dorsal or first lumbar vertebræ. In the dorsal and lumbar regions, the bodies are fractured in two thirds of the cases, but in the cervical region, the arches suffer alone in one half. Usually there is a displacement of the vertebral column at the seat of fracture. Rarely the bodies may be crushed without displacement. The displacement of the bone involves a narrowing of the canal and, usually, compression of the cord. This may also result when the arches only are driven in. But the cord may be seriously damaged when there is no permanent narrowing of the canal, as in Figs. 76 and 77. The dura mater is rarely torn except



FIG. 76.—Fracture of the first lumbar vertebra. D M. Dura mater. There was no permanent narrowing of the canal, but, nevertheless, the spinal cord was greatly damaged at the spot; see next figure. (From a drawing by Mr. V. Horsley.)

by a splinter. Blood is almost always extravasated outside the dura mater, often in considerable quantity, from the rupture of the large veins in this situation. There are usually only small extravasations in the pia mater. The cord is, in most cases, bruised and compressed by the lower fragment (Fig. 78). Sometimes it is flattened, and it may even be divided, all nerve-substance being squeezed out of the pia mater at the spot. In the case shown in Figs. 76 and 77, the cord appeared to have been split longitudinally at the spot. Blood is extravasated into the bruised part, sometimes in minute spots, sometimes in larger hæmorrhages, which may occupy longitudinal cavities. These changes are usually limited to the spot compressed. Secondary myelitis is set up in the damaged part, and this sometimes extends beyond the contused area. In cases of some duration the usual ascending and descending secondary degenerations are also found.

FIG. 77.

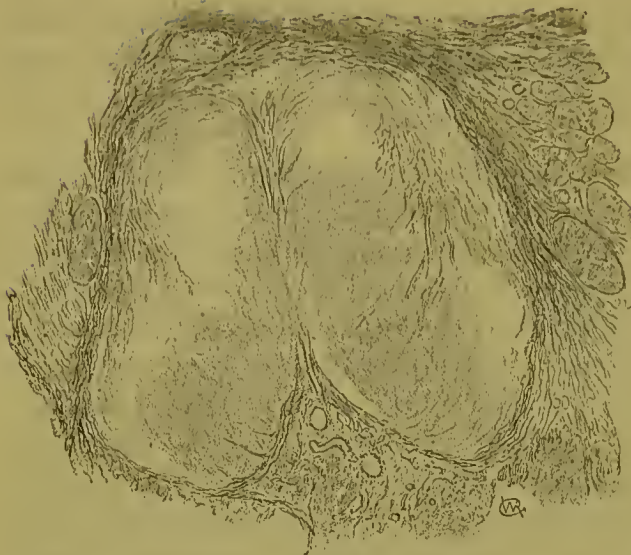


FIG. 78.

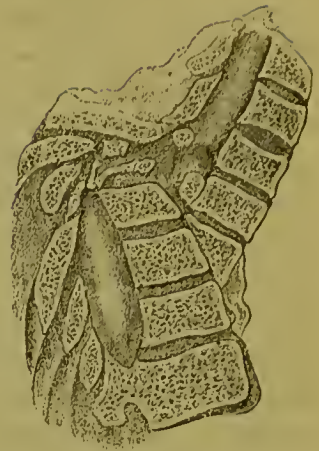


FIG. 77.—Spinal cord damaged by the fracture shown in Fig. 76. The elements of the cord itself are changed beyond the possibility of identification. The ascending degeneration is shown in Fig. 67.

FIG. 78.—Fracture of the body of the fifth dorsal vertebra and of its processes. (After Gurlt.)

Fig. 67, p. 117, represents sections of the cord from the case of fracture figured above. Occasionally the injury leads to secondary caries of the bone, with all its consequences.

Three classes of symptoms result. The first consists of the local indication of the injury. Secondly, there may be certain nervous symptoms, not distinctly due to the damage to the cord. One of these is general shock, which may be so great as to entail transient loss of consciousness. Vomiting occasionally occurs. There is great pain in the position of the fracture, rendered very intense by pressure, and often radiating along the nerves which come from this part, the roots of which are compressed. Tetanus occasionally ensues, and has been ascribed also to the irritation of the nerves. In rare cases epileptiform convulsions have followed fracture, usually at an interval of some days.

The third group of symptoms are those which result from the damage to the cord, and consist in paralysis of the parts below the injury. Its character depends on the amount of damage. If this is considerable, there is both motor and sensory paralysis up to the level of the lesion, with loss of power over the sphincters. Reflex action is lost at the level of the lesion, and the examination of the trunk-reflexes often gives important information regarding the extent of the damage, when this is in the dorsal region. Below, reflex action is commonly in excess, unless the centres are impaired by descending myelitis. Spasmodic twitchings sometimes occur in the limbs immediately after the injury, accompanied by priapism. The pains are severe in the arms when the fracture is opposite the cervical enlargement, and in the legs when at the lumbar enlargement, or below the latter, so as to damage the nerve-roots. In

these cases there may be rapid wasting of the muscles, with loss of electric irritability. There is usually at first incontinence, afterwards retention of urine, but the former is persistent if the lumbar centres are damaged. Cystitis, bedsores, &c., may supervene. Ultimately, if the damage is above the lumbar enlargement and the patient lives, there may be increased myotatic irritability in the limbs, increasing to spasm, so that spastic paraplegia results.

Special symptoms result when the injury is in certain parts of the spine. Fracture of the *first two cervical vertebræ* causes instant death, unless the displacement is very slight, and even then there is imminent danger of further displacement, with the most serious consequences, on any incautious voluntary movement. With slight displacement persons have been known to live for weeks, and then die from secondary myelitis; they have even recovered altogether. Now and then there is no compression of the cord, although there is distinct displacement, even sufficient to be recognised in the pharynx (Leyden). In such a case death has resulted at a later period from caries. The characteristic symptoms are local pain, increased by all movements (which are thus rendered impossible), displacement, and spinal symptoms. The latter may be slight—merely difficulty in breathing or swallowing—or considerable, and involving the trunk and limbs. Sometimes there is hyperpyrexia. Not more than one case in fifty recovers.

Middle Cervical Vertebræ.—The third, fourth, and fifth vertebræ are most frequently fractured. When the injury to the cord is considerable, death usually occurs very rapidly, because the roots of the phrenic nerve are involved. In some cases there is little immediate displacement and the symptoms are slight until further displacement occurs in some movement. Thus a man who had met with an injury of this kind went to be shaved; during the process, his head was turned on one side by the barber, with the unexpected result of causing displacement of the fracture, and immediate death. When the fracture is at the *cervico-dorsal* region, opposite the lower part of the cervical enlargement the arms frequently escape at first, the early paralysis being confined to the legs and muscles of the trunk. Respiration is diaphragmatic only. After a few days the arms become involved, but their paralysis is often partial, affecting, for instance, only certain muscles, as the extensors of the hand, and it is often accompanied by local spasmodic movements. Movements and pressure cause pain, and there is local muscular rigidity. The head may be in normal or in abnormal position. Vaso-motor disturbance in the face and general hyperpyrexia have been observed.

In fracture of the *dorsal vertebræ* (2-11) the arms escape, the legs are paralysed, and the trunk-muscles up to the height of the lesion. The pain in the trunk may be very severe. There is hyperæsthesia or anæsthesia in the parts below. The reflex action in the legs is excessive; that in the trunk is abolished at the level of the lesion. The *last dorsal* and *first lumbar* vertebræ are often fractured, and there results

paralysis of the legs, complete or irregular, severe pains, tingling, &c., sometimes followed by hyperæsthesia or loss of sensibility, and by rapid disturbance of nutrition, in both the muscles and the skin. In fracture of the *lower lumbar vertebræ* the symptoms are often slight; below the extremity of the cord the nerves occupy a smaller space in the canal, and so may escape compression by a moderate displacement. The fractured vertebræ unite very slowly. A false joint is occasionally formed. If the patient recovers from the immediate effects he often dies from secondary myelitis and its consequences.

CARIES OF THE SPINE.

Caries of the bones of the spine is a frequent cause of paraplegia. It is often termed "Pott's disease," from the English surgeon, Percival Pott, who first described it (in 1779) as a cause of paralysis.

CAUSES.—Males are said to be rather more liable than females, but the difference in sexual incidence is not great. It is more common in childhood (after three) and next in early adult life, but it may occur at any age, and is perhaps more common in the second half of life than any other scrofulous lesion. I have known it commence at fifty, and it has been met with as late as seventy. It is distinctly a manifestation of the tubercular and scrofulous diathesis, and evidence of such inheritance is to be traced in most cases. Occasionally the sufferer himself presents such indications, lung disease, &c., or caries of other bones. It occasionally develops simultaneously with other signs of acute tuberculosis. Caries has been thought to result from syphilis, but this cause is certainly rare.

Injuries seem frequently to excite the bone mischief in the spine, as they certainly do analogous bone disease elsewhere, in those who are predisposed, possibly, sometimes, in healthy persons. Falls, blows on the back, and severe strains, are the most frequent traumatic antecedents. The last may act by straining the ligaments, and setting up inflammation which spreads to the bones directly or through the intervertebral cartilages. There is usually an interval, sometimes of many months, between the injury and the definite symptoms of bone disease. Adjacent inflammation, secondarily affecting the bone, is a rare cause. Caries of the cervical spine has thus been secondary to a primary retropharyngeal abscess. The disease sometimes follows septicæmia in a way that suggests a secondary abscess in the bone. This was the case in a surgeon about sixty years of age, in whom symptoms of blood-poisoning followed an injury to the ankle. These were succeeded by paraplegia of rapid onset, and this by the development of angular curvature in the lower dorsal region. In a large number of cases no exciting cause can be traced.

PATHOLOGICAL ANATOMY.—The changes in the bones need not detain us, since they belong to surgical pathology, except in so far as they lead to damage to the cord. There is inflammation of the bodies of the vertebræ, and of the intervertebral substance, at first with enlargement, afterwards with breaking down of the substance of the bone, which gives way under the pressure to which it is exposed, so that deformity of the spine results. Pus often accumulates outside the spine, and may burrow in various directions, according to the seat of the disease.

The inflammation extends to the loose cellular and adipose tissue between the bone and the dura mater, and often to the dura mater itself, the outer layer of which becomes irregularly thickened in the neighbourhood of the disease (see Fig. 80). The inner surface of the dura mater is often normal when the outer surface is much changed. Inflammatory products, cheesy material, pus, &c., accumulate between the bone and the dura mater, and often in such quantity as to compress the cord. Inflammation is frequently set up in the cord, but usually only when it is also compressed.

The displacement that results from the collapse of the bodies varies much according to the extent and character of the disease. Occasionally there is simply a lateral displacement—one spine is situated a little to one side of that above it. More commonly the collapse of the bodies leads to “angular curvature,” the spinal column is bent forwards at an acute angle, and one or two vertebral spines are much more prominent than the others. There is often, however, a less abrupt bend; the curvature may extend over four or five vertebræ.

The *nerves*, as they pass through the membranes and intervertebral foramina, are irritated by the inflammation and often compressed by the thickening of the dura mater which sheaths them. Those passing by the seat of compression may also suffer from the narrowing of the canal. They may be found red and swollen, or shrunk and grey. They may be damaged when the cord is normal, or but little affected when this is compressed.

The damage to the *spinal cord* depends on the secondary consequences of the caries, and both its occurrence and character are variable and uncertain. The mechanism of the damage is twofold—compression and inflammation. The relation between the two is considered more fully in a subsequent chapter (“Compression of the Spinal Cord”). There is never compression without inflammation, but it is probable that, in many chronic cases, the inflammatory changes are secondary to the damage to the nerve-elements. On the other hand there may be inflammation when there is no compression. The mechanism of compression varies. The most frequent is the collection of inflammatory products outside the dura mater, and the thickening of this membrane. Less commonly the cord is compressed by the displacement of the bone, or by fragments of bone that are pushed into the canal. Often both these causes are influential, as in

the case shown in Fig. 79. Probably an inflammatory swelling or abscess of the bone is sometimes the mechanism of compression, since the signs of pressure have disappeared when an abscess has formed outside the spine, or even when deformity has come on. The inflammatory changes are most intense at the point of greatest compression, but extend, lessening in degree, through a few inches of the cord above and below the lesion. They are described in the chapter on "Compression."



FIG. 79.—Caries of the spine, mid-dorsal region. The spinal cord is much narrowed and discoloured at *s* from compression between the displaced bone and a mass of inflammatory products *E*, outside the dura mater, *DM*, the inner surface of which is normal. (After Leyden.)

SYMPTOMS.—Caries of the spine causes symptoms of three classes:—(1) those of the bone disease; (2) the effects of damage to the nerve-roots; (3) those due to the changes in the cord itself. Only the symptoms due to the damage to nerves and cord come within the special province of this book, but the bone symptoms are of much importance, as on them the diagnosis of the

nature of the nervous symptoms often depends.

These vertebral symptoms are various. The first is pain in the spine, chiefly felt at the affected spot, often increased by movement, and especially by pressure on the bone. The local tenderness is a very important sign. It is elicited not only by direct pressure on the spines, but by lateral pressure; if they are grasped and pressed to one side considerable pain is usually produced. The increase of pain by movement is greatest when the disease is in the more mobile parts of the vertebral column, especially when it is in the cervical region. Movement of the head occasions pain, and there is an instinctive fixation of the head, which is sometimes inclined to right or left, less commonly backwards. It may thus produce the aspect of torticollis, which differs from that due to muscular contraction in that the sterno-mastoid is tense on the side towards which the head is turned, the muscle being simply stretched by the deviation of the head.

The deformity of the spine is a later symptom than the tenderness, and usually comes on gradually. Its characters have been already described. It is often absent when the disease is in the cervical region. In this part there is usually another symptom, thickening of

the tissues about the spine. This is rare in other parts. In any region an abscess may form in the neighbourhood of the disease. Those which come backwards, or descend to the groin by the psoas muscle, can be recognised externally. Those that form in front of the diseased vertebræ may give rise to symptoms that are puzzling, if the existence of spinal caries is not known. Thus a retropharyngeal abscess may cause difficulty of deglutition, and one in the dorsal region may cause symptoms of œsophageal obstruction, &c.

Increased pain and tenderness in the vertebral column often precede the symptoms of damage to its contents. The symptoms due to interference with the *nerve-roots* are very variable and are often absent. They are both sensory and motor. There is pain along the course and in the distribution of the nerves, and hyperæsthesia of corresponding extent, sometimes with spots of anæsthesia. There is also muscular weakness, and sometimes muscular wasting. These symptoms are more fully described in the chapter on "Slow Compression." Here it may suffice to say that they are conspicuous only in a minority of the cases, and chiefly occur when there is pachymeningitis. The motor symptoms are most marked when the disease is in the cervical region, and the nerve-roots for the muscles of the arms are damaged, since slight and limited impairment is more readily recognised in the arms than in the trunk. If the disease is in the highest part of this region, the pains may be referred to the occiput. Herpes zoster has been occasionally met with along the course of the irritated nerves. Spasmodic contraction in the muscles supplied by the affected roots is extremely rare in cases of caries. Reflex action is abolished in the affected parts, and the change in the superficial reflexes of the trunk sometimes gives important diagnostic information. Disease of the lower cervical roots may cause symptoms of deranged action of the sympathetic on that side of the head, occasionally shown in the pupil, more often in the vessels. I have seen persistent sweating on one half of the forehead from this cause.

The symptoms of interference with the function of the spinal cord itself are chiefly impairment of its conducting power, causing paralysis in the parts below the lesion. As the disease is most frequently in some part of the dorsal region, paralysis of the legs is the common effect. But the symptoms vary in their characters, according not only to the position of the disease, but also to the immediate process of damage to the cord. The signs of caries may have existed for years before paralysis comes on. Angular curvature may even come on in early childhood, and paralysis not till adult life. More commonly the paralysis comes on one or two years after the curvature; sometimes at the end of a few months. On the other hand, the two classes of symptoms may manifest themselves at the same time, and the symptoms of cord disease may even develop, and reach a considerable degree, before the local symptoms are sufficiently marked for bone disease to be suspected. In one patient, after absolute paralysis of the

legs had existed for six months, an experienced surgeon doubted the existence of bone disease, but a few months later the signs of this were indubitable.

The cord symptoms vary much in their mode of onset. Usually there is no exciting cause, but sometimes a strain of the back or exposure to cold seems to excite a change in the condition of the bones, in consequence of which the cord suffers. It is easy to conceive that, when the conditions are favorable, a very slight strain may be effective. In one case, the first symptoms followed instantly three violent sneezes. When the cord symptoms have commenced, they may develop quickly or slowly. As instances of chronic onset may be mentioned cases in which the symptoms reached a considerable degree of intensity at the end of nine months, four months, and two months, after their commencement. But the onset is sometimes much more rapid; in one case there was complete paraplegia at the end of three weeks. Sometimes it is still more acute, and in such cases, myelitis rather than pressure is probably the effective agent. Thus, in one patient, a child of three, slight weakness existed for three weeks, and then the power of standing was lost in a single night. In another child, aged eight, who had presented, for two years, indications of disease of the cervical vertebræ, the power of moving the legs was lost in the course of twenty-four hours; during the second day the left arm became paralysed, and, at the end of a week, the right arm. Very rarely the onset is instantaneous, probably from sudden displacement, since hæmorrhage has not been proved to occur. A child with angular curvature was walking across the room, when she fell, and, on being lifted up, the legs were powerless.

Both legs usually suffer together; rarely one is paralysed before the other; very rarely one leg suffers much and the other little or not at all. Thus in one case angular curvature developed in childhood; at sixteen there was an attack of weakness in the legs, which passed away at the end of three weeks. At 17½ the patient sprained his back; pain in it followed, and six weeks later the right leg gradually became weak, and a year afterwards presented intense spastic paralysis, the left leg being very little affected. He ultimately recovered. An instance in which one leg became affected before the other is a case already alluded to. A woman forty-five years of age suffered from pain in the spine, and one day, when walking, sneezed three times, and immediately felt "pins and needles" in the right knee, and presently in the foot. The leg became almost powerless during the next three days. A fortnight afterwards, she felt similar tingling in the left knee, and, at the end of another week, in the foot, and during the next three weeks this also lost power, so that at the end of six weeks from the onset both legs were motionless. The diagnosis was verified on her death, six months later.

The characters of the paralysis in relation to the seat of the disease are described in the chapter on "Compression." In the most frequent

cases the dorsal cord is damaged and the condition of the legs is that of spastic paraplegia. If it is situated in the cervical region there is muscular atrophy in the arms, sometimes combined with palsy without atrophy, according as the disease is at the level of the cervical enlargement, or above this. There is the same spastic paralysis in the legs as when the disease is in the dorsal region. When, as is commonly the case, the arms suffer from damage to the nerve-roots, they suffer before the legs, but if the disease is so high up that the arms suffer from the damage to the cord itself, the legs may be paralysed before the arms, as in the case of the child mentioned above. In this case the muscles of the shoulder were wasted, the disease being near the level of their nerves, while the forearm muscles were not wasted. In this case, as in others of similar seat, the diaphragm was paralysed. The muscular part of the spinal accessory may be involved, and the power of supporting the head may be almost lost. Very rarely, from disease of the highest cervical vertebræ, the other nerves of the medulla are implicated.

Sensory symptoms due to the disease of the cord are less common than motor palsy. As a case already mentioned shows, the onset of paraplegia may be heralded by subjective sensations in the legs. Dull aching pain in them is not uncommon. Often there is no anæsthesia; in other cases there are various degrees of loss of sensation. Touch or pain may be lost alone, or there may be absolute loss of sensibility up to the level of the lesion. Reflex action in all forms is excessive (unless the disease involves the lumbar enlargement), and great excess of the superficial reflex action is a common and important feature. The legs are often cold and sometimes perspire continually. The sphincters are often affected, sometimes early, but they may escape even when there is a complete motor palsy of the legs.

Complications.—Among common complications are bedsores, cystitis, various secondary effects of the bone disease and general tuberculosis. In very severe cases peculiar secondary mischief has occurred in the spinal cord, and has run an independent course, giving rise to very anomalous symptoms.* Thus a descending myelitis may invade the lumbar enlargement in its entirety and abolish its central and reflex functions, causing rapid wasting of the muscles and acute trophic changes in the skin. Inflammation may *ascend* the pyramidal tracts and thus paralyse the arms. Ascending degeneration of the posterior median columns may spread to the postero-external columns and cause symptoms of ataxy. It is probable also that a similar affection of these columns may descend the cord, since ataxy may come on as the power returns, when the disease is in the dorsal region. Lastly, myelitis may occur in disseminated foci in various parts of the cord, and even in the medulla, giving rise to scattered symptoms of anomalous character.

Course.—The bone disease may heal, ankylosis occurring, or it may persist with continued formation of pus, or it may become quiescent

* See Charcot, 'Leçons sur les Mal. de Syst. Nerv.,' tom. ii.

and from time to time become active. The cord mischief is influenced by the state of the bone disease, although its progress may be to some extent independent. Thus inflammation in the cord may subside in spite of the progress of that in the bone. Pressure on the cord may be relieved, although the bone disease continues, and even sometimes as a result of the increased breaking-down of bone and exit of pus by another channel. Hence there is no strict correspondence between the course of the bone mischief and that of the cord disease. In some cases the paralysis, motor and sensory, remains absolute. More often the sensory loss passes away, while motor paralysis remains, usually as spastic paraplegia, with muscular contractions due to posture. Life may be prolonged in that condition for years, but often bedsores form, or cystitis leads to kidney disease, and thus the patient dies, or the lessened respiratory power renders an attack of bronchitis fatal. In many cases, again, the paralysis gradually passes away even when the compression continues, and the cord is found considerably narrowed if the patient dies from some other cause. In children recovery occurs far more readily than in adults. Even in adults, however, recovery may occur from palsy that has lasted for some years with all the signs of descending degeneration in the cord. Usually rest on the back or mechanical supports are necessary to secure recovery, but it occasionally occurs without these measures. A youth acquired angular curvature at sixteen; at twenty paraplegia came on slowly, and progressed, with some variations, during the next seven years. There was then absolute motor palsy in the legs, and sensation was lessened. He continued to follow his occupation, a tailor, but took cod-liver oil and iron, and he gradually regained useful power, so as to be able to walk about. Such a case, however, is exceptional.

Relapses are apt to occur in cases that improve, although they are certainly not nearly so frequent as might be expected from the nature of the disease. In the majority of cases recovery is permanent. In a minority the paralysis returns when some exciting cause renews activity in the bone disease. The tendency to relapse and possibility of repeated recovery are very strikingly shown by the following case. In a girl of fifteen, paraplegia developed during nine months, slowly at first, more rapidly towards the end of that time. She came under my care six months later, having been unable to walk for eight months, to stand for seven, and to move her legs for six. Bone disease had not been previously suspected, but there was slight tenderness and enough lateral irregularity of the mid-dorsal spines to show the nature of the case. Rest in bed and tonics were soon followed by improvement; in six weeks she could stand, and even walk with help, and in four months was discharged, able to walk well. As she gained power, angular curvature came on, prominence of the seventh and eighth dorsal spines. Five months after her discharge, she fell and struck her back. The curvature increased, and her legs gradually became weak again. Seven months after the fall she was readmitted,

unable to stand, although the paralysis was not absolute. There was foot-clonus on each side. Sensibility was lessened below the ensiform cartilage. Rest on the back was again followed by slow improvement. In three months she could just walk. She was then suspended, and encased in plaster of Paris. At the end of another month she could walk about the room, and no clonus could be obtained. She was soon afterwards discharged, and her progress continued, so that, at the end of nine months, she could walk five miles, and there was no trace of clonus, although there was still some excess of the knee-jerk. She soon afterwards married, and bore a child, which died two years and a half after her discharge. She caught cold at the funeral, and a fortnight later again began to lose power; in six weeks the legs were almost motionless, with marked foot-clonus; sensation to touch was lost up to the umbilicus, that of pain being preserved. Neither rest nor encasement caused any improvement. After some months sulphide of calcium was given, and in a few days power began to return; in a month she could take a few steps, and in four months she could walk about the ward without difficulty. She made another good recovery. Some years later, however, paralysis again came on, and this attack proved permanent.

DIAGNOSIS.—When clear indications of caries precede the paralysis, the nature of the case can hardly be mistaken. The obvious inference, that the affection of the cord is secondary to that of the bone, is scarcely ever wrong. When the two develop together, mistakes in diagnosis are often made, but are usually due to the want of *repeated* examinations of the spinal column. It is when the root or cord symptoms precede distinct evidence of bone disease that the chief real difficulty in diagnosis occurs; the affection is apt to be mistaken for a primary disease of the cord or its membranes, a transverse myelitis when the dorsal region is affected, a progressive muscular atrophy, or primary pachymeningitis, when the disease is in the cervical region. A correct diagnosis can only be made in these cases by recognising the significance of the slight bone symptoms that are always present, the deep tenderness and often slight irregularity. Even slight irregularity derives significance from tenderness, limited in extent, and corresponding in position to the deviation. The irregularity may be absent at first, and then its development is doubly significant. So, too, an increase in the amount of displacement may give significance to an irregularity that is no greater than is occasionally met with in normal spines.

The early excess of the cutaneous reflex action (from the sole, for instance), while not diagnostic, adds weight to the other symptoms of bone disease. It may be very marked, even while the patient is able to walk about. When root-pains are present of definite character, they give additional significance to the other symptoms, since it is rare for a primary myelitis to cause more than a sense of constriction. Spots of anæsthesia along the course of the nerves are of still greater

diagnostic value. All these symptoms derive additional weight from their coincidence in level with the deep spinal tenderness. The anæsthesia, with hyperæsthesia and bone tenderness, sometimes serves to distinguish the pains from those of intercostal neuralgia, with which they may be confounded at an early stage of the disease.

When damage to the cervical roots causes muscular wasting in the arms, the case may be mistaken for one of progressive muscular atrophy. The irregular distribution of the wasting differs from that common in the latter disease, and in this there are none of the sensory symptoms which are usually so pronounced in caries. The same symptoms may, however, be produced by a primary pachymeningitis, and the further distinction between the two rests on the presence of the signs of bone disease, on the absence of the causes of meningitis (of which syphilis is the most common), and on the youthful age of the patient; cervical symptoms are more likely to be due to caries if the patient is young than if he has reached adult life, and conversely they are more likely to be due to a primary pachymeningitis in adult life than in youth.

In the first half of life the recognition of bone disease is practically tantamount to the recognition of caries. In the second half, however, the relative infrequency of caries, the greater frequency of growths in the bone, and the occurrence of eroding aneurisms introduce a fresh diagnostic problem. The absence of any other indication of a tumour or an aneurism is the first distinction, and the second is the fact that in both these diseases the root pains commonly reach a degree of severity scarcely ever attained in caries, and are especially increased by movement, often to an intensity which is literally frightful.

When there are merely tenderness of the spine and slight weakness of the legs, the question may arise whether there is organic disease, or merely the condition termed "spinal irritation." In the latter, tenderness is usually found over a considerable area of the spinal column, with more than one point of special intensity, and there are no root pains or spots of anæsthesia. The latter sometimes afford important help in the diagnosis of an obscure case. In some instances, however, it is necessary to wait and watch the progress of the symptoms before a definite opinion can be formed. The same general principles must guide the diagnosis between caries and so-called hysterical paraplegia with spinal tenderness. There is more danger that caries of the spine in a young woman may be passed as hysterical paraplegia, than of the opposite error. Especially when the subjects of caries present distinct symptoms of hysteria, there is risk, as experience shows, that unequivocal symptoms of caries may be overlooked.

PROGNOSIS.—Our ignorance of the precise character of the morbid process which is damaging the cord renders the prognosis, in every case of caries, a matter of much uncertainty. Nevertheless there is

no disease of the cord in which symptoms of equal gravity so often pass away. The cases are few, therefore, in which hope is unjustified, but they are equally few in which we are warranted in a confident expectation of recovery. The indications that may guide the prognosis in an individual case are slight. In childhood the prospects of recovery are certainly better than in adult life, and they are least in declining years. Damage to the cord between the enlargements is less serious than when these are affected, because the strong tendency to trophic changes constitutes a grave danger when the lumbar enlargement is diseased, and the diminished breathing power an equally serious danger in disease of the cervical enlargement.

But how perilous a condition may be recovered from is shown by the fact that the child mentioned on p. 172 with paralysis of all four limbs, the diaphragm, and weakening of the intercostals, made a good recovery. Still more striking is a case narrated by Dr. Buzzard, in which disease in the region of the third cervical vertebra caused almost complete palsy of arms, legs, intercostals, and diaphragm, respiration being carried on by the accessory muscles of the neck. Yet the child recovered in spite of the occurrence of an attack of pneumonia when the paralysis was at its height. Neither rapidity nor slowness of onset affords any guide to prognosis, nor does the relative order of paralysis and curvature, nor the degree of palsy. Even pronounced spastic paraplegia may pass away entirely. The prognosis is perhaps a little better when there is no loss of sensation, since this proves that the damage to the cord is moderate in degree, but even complete anæsthesia does not preclude recovery, as the cases mentioned show. The danger to life is dependent in considerable degree on the evidence that the scrofulous or tubercular tendency is active elsewhere, and also on the possibility of securing proper treatment.

TREATMENT.—The first and chief element in the treatment of paralysis is that of the bone disease which causes it, and for full details of this the reader is referred to treatises on Surgery. If the bone disease heals, the spinal cord, in most cases, will recover. The two most potent therapeutic agents are the customary measures,—persistent recumbency, and tonics, especially cod-liver oil and iron. In the cases that have done best under my own observation, these, and these alone, have been the effective agents. Rest should be maintained for months. The posture that answers best is upon the back. It is true that the spine is then the lowest part, and spinal congestion is favoured, but experience is here at variance with theory, and the greater ease with which immobility is secured and maintained seems to more than counterbalance the disadvantages of this posture. If no improvement occurs after some months' rest, suspension may be tried. It is said to be sometimes attended by instant improvement, especially in children. I have not myself seen any instance of this result, which can only occur in the cases, not very common, in which displaced bone compresses the

cord. It may, indeed, be well to see the effect of suspension before rest is commenced. The plaster-jacket is an inefficient substitute for rest, and an unnecessary concomitant—to be adopted only when rest cannot be secured. It may be tried if rest alone has failed. In caries of the mobile cervical spine, the fixation of the head by the support termed a “jury mast” is necessary.

The influence of cod-liver oil and iron is as marked in this as in other serofulous diseases. They constitute an indispensable adjunct to rest, and may be effective even alone, as is shown by the case mentioned on p. 174. But rest should always be secured if possible.

Counter-irritation, opposite the seat of caries, has often appeared to do good, perhaps acting especially on the process of myelitis which attends compression. The actual cautery, in mild form, is that which has been most frequently useful. Chareot and others have published instances of speedy improvement after its use. It is not always effective, nor can indications for its use be laid down. I have not, myself, seen any distinct improvement after the use of the cautery, although I have employed it in a considerable number of cases. But the testimony of others is so strong as to justify its adoption if the symptoms do not speedily yield to other treatment. Sulphide of calcium, which is said to influence serofulous processes, deserves trial. Although I have not seen benefit from its use in any other case than that mentioned on p. 175, the rapidity of improvement in that case was very striking. Benefit can only be expected from its use when inflammatory products constitute the compressing agent. Extreme care in general management, to avoid bedsores, bladder trouble, and bronchitis are of great importance. In many cases there is a tendency to improvement after a time, especially when the bone disease is stationary, or the cord has been inflamed, and in these the mere maintenance of life may result in recovery of strength. When power has returned, but the use of the limbs is restrained by the muscular contractions which came on during the paralysis, the ability to stand and walk may, as I have seen, be quickly restored by tenotomy, and more slowly by the use of extending splints. Electricity is useful only to maintain the nutrition of the muscles when these are wasting. The combined teaching of the pathology of caries and of the recent experience of antiseptic surgery justifies the anticipation that the trephine may sometimes be successful when other agents fail, since the cause of compression is generally outside the dura mater, and the power of recovery of the cord, when compression is removed, is very great.

TUMOURS AND OTHER DISEASES OF THE SPINE.

GROWTHS IN THE SPINAL COLUMN.

The bones of the spine are sometimes the seat of primary or secondary growths, and, less commonly, are invaded by tumours springing from the structures in front of the spine. Cancer (scirrhus and encephaloid) and sarcoma, are the most common forms of tumour that begins in or invades the spine. Myxoma has been also met with. Cancer is often secondary to a primary growth elsewhere, in the breast, stomach, &c. A primary growth usually begins in the bodies of the vertebræ, and spreads from one to another. The bodies may collapse, because the soft tissue which replaces the bone yields under the weight it has to bear. Thus angular curvature may occur. From the bodies the growth may extend into the lateral processes, enlarging them, and narrowing the intervertebral foramina through which the nerves pass. It may extend into the arches, and even into the spines, and may grow into the muscles and beside and behind the vertebral column. The nerve-roots suffer (1) by pressure, (2) by simple inflammation, (3) sometimes by cancerous infiltration. They may thus be found reddened, swollen and soft, or grey and atrophied, or enlarged and hardened. The growth, cancer especially, is apt to spread in the adipose tissue between the bodies and the dura mater. It may even entirely surround the dura mater and cord. The cord may suffer compression, but this is less frequent than in caries. On the other hand, it is often inflamed, sometimes acutely, and considerable inflammation without compression is far more frequent than in caries. Myelitis occurs without any perforation of the dura mater. Why this should be is not known. The inflammation thus excited sometimes spreads widely in the cord. The growth never invades the cord itself. In rare cases the cord is compressed by displaced bone.

ETIOLOGY.—The general causes are the same as those of similar growths elsewhere. Males suffer, however, more frequently than females, and the disease is most common between forty and fifty years of age. An injury has been supposed sometimes to excite a growth in this as in other situations.

SYMPTOMS.—There may be direct symptoms of the presence of the growth—pain in the spine, sometimes severe, sometimes absent; local tenderness, and occasional interference with movement apart from pain. A palpable tumour is never an early symptom, but ultimately a deep-seated hard swelling may be felt on one side of the spine, most readily and earliest when the disease is in the cervical region, where it is occasionally perceptible in the posterior triangle of the neck. The angular curvature which sometimes occurs may not differ from that of caries in its external characters, or it may be accompanied by the indications of a tumour, never by those of an abscess.

The nerves suffer almost invariably in spinal growths and give rise to the most distressing symptoms, which are also the earliest and most frequent,—radiating pain, felt along the course of the nerves that emerge from the diseased portion of the spine, and due to their irritation by pressure or inflammation.

Such pains are far more prominent symptoms in this disease than in caries. At first slight, they gradually increase to extreme intensity. It was this character that led Cruveilhier to call the disease *paraplegia dolorosa*, a name it has since commonly borne. The pains are paroxysmal; in the intervals the patient is at first free from pain, but subsequently pain is constant, with paroxysms of greater suffering from time to time. It is usually sharp, lancinating pain, and its special characteristic is the degree in which it is increased by movement. The distribution in the arms, trunk, legs, depends on the seat of the disease. It is extremely rare for these pains to be absent, but they are now and then a late, instead of an early, symptom.* Cutaneous hyperæsthesia usually accompanies them, and spots of anæsthesia often develop after a time in the hyperæsthetic area. Corresponding damage to the motor roots may cause painful muscular contracture (Fig. 50, p. 78), paralysis, and wasting. Paroxysms of spasm often attend the pains, especially in the abdominal muscles when the disease is in the dorsal region, and are apparently reflex in nature.

The damage to the cord causes symptoms similar to those in caries, and described more fully in the chapter on "Compression." The chief difference from caries is the frequency of a rapid onset of the paraplegic symptoms, due to the invasion of the cord by acute inflammation. All power in the legs is often lost in twelve or twenty-four hours from the onset, and this when no curvature has taken place. Displacement of bone has also been known to cause rapid paralysis.† On the other hand, in very slowly growing tumours, the onset of the palsy may be gradual. I have known it to occupy several years in reaching a considerable degree. Probably in these cases the mechanism is a pure compression. Thus, compared with caries, the onset is more often rapid, and occasionally much more deliberate. The characters of the resulting paralysis are, as a rule, similar to those in caries, but are more frequently modified by the spread of inflammation. Hence the central and reflex functions of the lumbar enlargement, at first normal or excessive, may be afterwards lost, although the bone disease is some distance above it. In a man with a growth in the mid-dorsal region, paraplegia came on rapidly, evidently from myelitis, and was followed by foot-clonus, &c. But a few weeks later the clonus suddenly ceased, the muscles became toneless, with loss of faradaic irritability, and the skin began to slough. The inflammation had spread down into the lumbar enlargement. Sensation is lost rather more frequently

* The pains succeeded curvature in a case described by L. Humphry ('Lancet,' 1884, i, p. 15).

† Humphry, loc. cit.

than in caries. Other symptoms are the same as in compression from any cause. The course of the disease is, from its nature, usually progressive. Occasionally, however, some improvement occurs. An inflamed cord may partially recover if life is prolonged for a sufficient length of time. Much more rarely the pains may lessen, although the growth spreads—perhaps from destruction of the irritated nerves.

The *duration* of the disease varies according to the nature of the growth. In cancer it may almost always be measured by months. In slowly growing tumours the symptoms may last for years. Death may be due to bedsores, &c., to cystitis and kidney disease, to growths elsewhere, or the patient may be simply worn out by the prolonged agony.

DIAGNOSIS.—The recognition of the disease is only a matter of certainty when signs of a tumour are present, but the probability almost amounts to certainty when such symptoms as those described follow a primary growth elsewhere. It must be remembered, however, that similar root symptoms are sometimes due to a growth in front of the vertebral column, commencing, for instance, in the glands, and irritating the nerves as they emerge from the intervertebral foramina.

From intercostal neuralgia, the influence of movement on the pain is usually a sufficient distinction, even when cord symptoms are absent. The commonly bilateral character of the pain is a further difference. The chief difficulty in diagnosis is the distinction from caries when other evidence of growth is absent. In the first half of life caries would alone be thought of in such a case, but in the second half the two diseases are about equally frequent. One distinction, suggestive, not absolute, is the intensity of the pain in tumour, taken in conjunction with its great increase when the patient moves. It is true that the root pains of caries are said to be sometimes most severe, but such severity is not frequent enough to destroy the significance of intensity. I have not, for instance, seen a single case of caries (of a large number) in which the pain was comparable to that in most of the cases of growth that have come under my observation. Therefore, while absence of pain is of slighter diagnostic value (in favour of caries), severe intensity, and agonising increase by movement, are strongly in favour of vertebral growth. In each disease there may be angular curvature, but this, in growths, is usually soon succeeded by other signs of tumour. In caries these signs are absent, and an abscess often develops. A history or indication of tubercle is strong evidence in favour of caries. These points will, I believe, avail for the distinction in most cases. In a few it is necessary to wait and watch before an opinion can be formed. The distinction from tumours of the spinal cord and membranes is considered in a later chapter.

PROGNOSIS.—The prognosis scarcely requires formulating. The chief differences are in the time that life is likely to last. The pains usually persist, in spite of the progress of the disease, although there is a bare possibility of their subsidence. The chance of any return of

power in the paralysed part is small, although not quite absent if the palsy develops in a manner to suggest a secondary myelitis, and the progress of the growth itself is slow.

TREATMENT.—This must necessarily be confined to the relief of pain, and to the avoidance of bedsores and other results of the cord disease. Morphia is alone effective for the relief of the pain, but unhappily the dose has to be quickly increased, and the power of the drug is lessened by custom. It becomes a race between dose and pain, in which, if life lasts long, the pain not uncommonly gets in front of the narcotic. It remains to be seen whether, in this as in so many other diseases, cocain may prove an effective substitute for morphia.

VERTEBRAL EXOSTOSES.

Exostoses sometimes grow from the bodies of the vertebræ into the spinal canal, and may compress the cord or nerves. They are, however, exceedingly rare. The symptoms may be those of slow compression of the cord, or of irritation, expressed chiefly by pain. They usually resemble those of a tumour of the cord or membranes rather than of the bones, but the pain is occasionally increased by movement. Their chief characteristic is extreme chronicity, but in this they are rivalled occasionally by some cord tumours. In a case (seen with Mr. W. Adams, of Camden Town) the patient suffered frequent intense paroxysms of pain in the right groin, which had occurred for two years, with occasional intervals of freedom. There was some weakness of the legs, but no considerable paralysis. Ten years before he had had some loss of sensibility in each thigh, which had passed away. An intraspinal tumour was diagnosed; the post-mortem examination (by Mr. Coode Adams) revealed exostoses from the bodies of the ninth and tenth dorsal vertebræ, slightly compressing the cord. Although extreme chronicity may raise a suspicion of exostosis it is doubtful whether a confident diagnosis is ever justified except in the cases in which there are similar exostoses elsewhere.

A patient with multiple exostoses, under the care of my colleague Dr. Barlow, presented paraplegia of gradual onset, which was supposed during life, and found after death, to be due to a similar exostosis within the spinal canal. It had sprung from one of the lumbar vertebræ and had compressed the nerves of the cauda equina.

SYPHILITIC DISEASE.

Syphilitic caries of the bodies of the vertebræ is a rare variety, the symptoms of which do not differ from those of the scrofulous form. It has been observed in the cervical region, secondary to deep syphilitic ulceration of the pharynx. Nodes of the vertebræ, within the canal, are occasionally presumed to exist and to compress the cord, but I do

not know of any pathological observation confirming the assumption and it is probable that most of the supposed instances have been cases of syphilitic gummata in the meninges. Deep-seated thickening of the tissues about the cervical vertebræ sometimes occurs in syphilitic subjects. It may develop on one side or both, and is apparently due to a syphilitic cellulitis. The swelling may be felt either on each side and behind the upper cervical spine, or deep in the posterior triangle of the neck. Movements of the neck may be interfered with, and irritation of the nerves may cause neuralgia-like pain, sometimes felt down the arm. The spinal cord does not usually suffer. All the symptoms slowly pass away if iodide is given.

EROSION BY ANEURISM.

Bones, like other structures, may atrophy and waste before the pressure of an aneurism, and the bodies of the dorsal, or rarely, of the lumbar vertebræ may be thus eroded by aneurisms of the aorta. The pressure and absorption take place from the left side. Two or three vertebræ usually suffer, and the bodies more than the intervertebral cartilages. The periosteum becomes thickened and may resist the pressure and to some extent protect the cord. Sometimes, however, the cord becomes compressed, or the periosteum may come to form part of the wall of the aneurism, and may give way before the blood-pressure so that rupture occurs into the spinal canal.

The symptoms vary much. Pain along the nerve-roots is usually severe, but this may attend aneurisms that merely compress the nerves after their emergence, and do not damage the bone. The process of erosion is usually attended by severe pain in the spine. When the cord is reached, compression causes the usual paraplegic symptoms, of slow or rapid onset. Rupture into the canal is attended by sudden complete paraplegia and death, either immediate, or in the course of a few hours from ascending paralysis, due to the hæmorrhage around the spinal cord.

The diagnosis is scarcely possible unless other indications of aneurism are detected, since the symptoms closely resemble those of a growth in the bone. The nature of the disease may, however, be suspected if such symptoms as have been described are succeeded by sudden paraplegia followed quickly by ascending paralysis.

HYDATID DISEASE.

Hydatid cysts sometimes develop in the loose adipose tissue between the dura mater and the bone, and it is believed that they sometimes form in the substance of the bones themselves. About a dozen cases have been collected by Leyden.* As the cyst

* 'Klinik der Rückenm. Krankh.,' Band i.

grows, the bone of the arches becomes atrophied by pressure, and the cyst develops outside the canal, so that there may be a double cyst, outside and inside, connected by a narrower part. Occasionally the cyst develops also in front of the spine, in the thorax or abdomen. The internal cyst necessarily compresses the spinal cord, which often also becomes inflamed. The usual paraplegic symptoms develop, both motion and sensation being often lost. Radiating pains along the nerve-roots are frequent. The symptoms, in themselves, resemble too closely those caused by other diseases of the spinal column to permit a diagnosis to be made unless similar disease elsewhere suggests the nature of the spinal lesion, or unless the cyst can be felt in the back, when a puncture may prove its nature. All the cases hitherto recorded have terminated fatally, but it has been conjectured that, if puncture can be effected, a cure may sometimes be possible.

DISEASES OF THE ARTICULATIONS.

LATERAL CURVATURE OF THE SPINE scarcely ever affects the functions of the cord. Even when slight compression has occurred, the slowness of its development has apparently prevented interference with function. In very rare cases, some weakness of the legs has been present, possibly, but not certainly, the result of the curvature. Occasionally the intervertebral foramina have been narrowed, and the pressure on the nerves has caused radiating neuralgic pains.

ARTHRITIS DEFORMANS.—In chronic rheumatoid arthritis, the intervertebral articulations may be attacked, and the cartilages may undergo atrophy. Enlargement of the ends of the bones may develop, and, in thin persons, the swelling may even be felt. The symptoms are local pain, tenderness, and limitation of movement, which may go on to absolute fixation, especially in the cervical region, if ankylosis occurs. The cord is scarcely ever compressed, but the narrowing of the foramina may damage the nerve-roots, and cause radiating pains, and sometimes set up a descending neuritis. The nerve-trunks in the limbs may then become tender as well as painful, muscular atrophy may occur, and, when the disease is in the cervical region, there may be symptoms of disturbance of the sympathetic. Spasmodic torticollis is said to have been caused by this disease in the cervical region. The atlo-occipital articulation has been affected alone, or with the adjacent vertebræ, and peculiar interference with the movement of the head has resulted. Sometimes the foramen magnum is narrowed, or the odontoid process projects into the canal. Symptoms of damage to the nerves of the medulla have been noted (Solbrig), but often the disease has been found out at the post-mortem examination without being suspected during life. The treatment of vertebral arthritis is the same as that of the disease elsewhere, nerve symptoms alone needing special

treatment, such as is described in the section on diseases of the nerves.

DISEASES OF THE MEMBRANES OF THE SPINAL CORD.

The general arrangement of the membranes of the spinal cord has been already mentioned (p. 104). The pia mater closely invests the cord, while the arachnoid forms a loose sheath around it, and the two membranes are connected by trabeculæ and membranous expansions of fine connective tissue, which occupy the "subarachnoid space." Each surface of the dura mater is covered by a layer of epithelium. The outer sheaths of the nerves are continuous with the dura mater; the connective tissue, within the outer sheath, is continuous with both the pia mater and arachnoid. The blood-vessels of the cord ramify, as we have seen, in the pia mater, and the lymphatic canals of the two are continuous. Most of the cerebro-spinal fluid within the vertebral canal is contained in the subarachnoid space, but there is a little fluid between the dura mater and the arachnoid. Both dura mater and pia mater are supplied with nerves, but those of the pia mater are the more abundant.

The morbid processes that involve the membranes are chiefly three, growths, hæmorrhage, and inflammation. The consideration of tumours of the membranes may be conveniently postponed until the consideration of growths in the spinal cord, with which they have much in common. Inflammation of the membranes and hæmorrhage into the membranes will be here described.

SPINAL MENINGITIS: INFLAMMATION OF THE MEMBRANES OF THE SPINAL CORD.

Inflammation of the membranes may be acute or chronic, and may begin, and affect chiefly, the dura mater (*pachymeningitis*), or the pia mater (*leptomeningitis* of German and French writers). The arachnoid usually suffers with the pia mater, but is sometimes the seat of inflammation that affects the pia mater but little, a form that has been termed *arachnitis*. Acute inflammation, beginning in one membrane, usually spreads to the others. Chronic inflammation may remain limited to one membrane, dura mater or pia mater. Inflammation may affect the outer or inner surface of the dura mater. All forms of acute inflammation, wherever they begin, cause similar symptoms. It is only when the inflammation is chronic that the symptoms may

differ sufficiently to allow of a precise diagnosis of the membrane affected.

Spinal meningitis may be conveniently divided into two forms: (1) that which begins outside the dura mater, and commonly arises by extension from some adjacent focus of inflammation,—*external meningitis*; (2) that which begins within the dura mater sheath, and is often primary,—*internal meningitis*. External meningitis may spread through the dura mater, and the two forms then coexist. Internal inflammation rarely spreads to the outer surface of the dura mater.

EXTERNAL MENINGITIS.

External meningitis is thus an inflammation of the dura mater. It has been termed *external pachymeningitis*, *peripachymeningitis*, and *perimeningitis*. The inflammation involves not only the outer surface of the dura mater, but also the connective tissue which intervenes between the membrane and the bones, and in which are the venous plexuses and a quantity of adipose tissue. In most cases this connective tissue is inflamed before the membrane itself. The morbid process usually remains limited to the outer surface of the dura mater, passing through the membrane only when the inflammation is acute and intense.

CAUSES.—It is doubtful whether external meningitis is ever primary. In most cases, perhaps in all, it arises by extension from adjacent disease. The most common cause to which it is secondary is caries of the spine. It may also result from deep sacral bedsores, and suppuration outside the spinal column, such as retropharyngeal abscess, or collections of pus in the muscles near the spine, in the back or abdomen. At the same time it is possible that an abscess found outside the spine has occasionally been secondary to the suppuration within the spinal canal, rather than the cause of this. External meningitis has been thought, in rare cases, to be due to extension from inflammation of the loose connective tissue beneath an inflamed pleura, to an ascending neuritis from a limb, and to syphilis.

PATHOLOGICAL ANATOMY.—The inflammation is sometimes simple, but far more frequently purulent, or semi-purulent. In the former case the outer surface of the dura mater may be merely reddened and opaque, with a little lymph on the surface. When the inflammation is purulent the surface may be covered by a layer of pus. Such inflammation is commonly the result of acute septic suppuration in the vicinity, and then it may pass through the dura mater, and purulent internal meningitis coexists. In the more common semi-purulent form, such as is common in caries of the spine, a layer of inflammatory

products covers the outer surface of the dura mater, soft, semi-caseous or firm caseous material, sometimes with liquid pus here and there, or contained in cavities within the firmer substance. This layer may be of considerable thickness, sometimes half an inch thick (see Fig. 79, p. 170). It may surround the dura mater, or may be chiefly situated on one side. Sometimes there is an irregular nodular thickening of the outer layer of the dura mater or even a sort of fungoid growth from it. An example of this is shown in Fig. 80. The bulk of these inflammatory products may cause serious compression of the cord. In most cases the inner surface of the dura mater and the pia-arachnoid are normal even when the cord itself is the seat of pressure-myelitis. Occasionally the inner surface of the dura mater is also inflamed, and adhesions may form between it and the pia mater. In all forms of external inflammation, the fat outside the dura mater quickly becomes absorbed. The membrane may ultimately become adherent to the bone. The vertical extent of the disease varies much. When secondary to caries, the inflammation is usually limited to the neighbourhood of the bone disease. In other cases the mischief spreads more widely, but when it begins in the dorsal or lumbar regions, it commonly ceases at the cervical region, in consequence of the closer attachment of the dura mater to the bone. In most forms of external meningitis the nerve-roots suffer as they pass through the membrane. They may be inflamed and swollen, or compressed and wasted.



FIG. 80.—External pachymeningitis; nodular thickening of outer layer of dura mater in caries of the spine.

SYMPTOMS.—In different cases of external meningitis the symptoms vary much, and they are usually complicated with those due to the cause of the inflammation. Those that are produced by the local inflammation which is secondary to caries have been already described in the account of that disease. In other acute cases, the manifestations of the process are nearly the same as those of internal meningitis, presently to be described in detail. The chief are pain in the back, increased by movement, accompanied by stiffness of the muscles of the spine, and by cutaneous hyperæsthesia. Another important symptom is the “excentric” pain, radiating into the parts supplied by the nerves which pass through the inflamed membrane, and due to irritation of their roots. The pains accompany the hyperæsthesia, and are usually associated with spasm; they may be followed by anæsthesia, and some-

times by paralysis of the muscles supplied by the nerves. In addition there may be indications of interference with the functions of the cord itself, due to its compression or inflammation. There are then pains in the limbs, often so great as to prevent walking, paraplegic weakness, and increased reflex action when the disease is above the lumbar enlargement. The skin of the limbs may be hyperæsthetic or anæsthetic, and trophic lesions may occur.

These symptoms differ in character according to the acuteness of the inflammation, and in distribution according to its seat. It is in the most acute form that the cord suffers and corresponding symptoms are developed. In such cases there is usually much pyrexia. In chronic cases, although the pain may be considerable, other signs of irritation, stiffness and muscular spasm, may be inconspicuous, and the symptoms resemble those due to bone disease.

DIAGNOSIS.—The most important indications of the disease are the vertebral pain, and the radiating pains, with other symptoms of irritation of the nerve-roots, combined with the signs of pressure on the cord itself. These symptoms have much in common with those of internal meningitis, and it is doubtful whether a diagnosis of external inflammation is justified, unless there is evidence of a morbid process to which the meningitis may be secondary.

PROGNOSIS.—The acute affection is a grave one, most recorded cases having ended in death. But it must be remembered that the disease is usually secondary to maladies which are in themselves most serious, and that the exact seat of the inflammation has usually been made out only after death. It is possible therefore that some cases which have recovered, and in which the exact form of meningitis was uncertain, may have been instances of this variety, and that the fatality of the disease may not be so great as published facts suggest. The chronic form, which results from caries, is only serious in the compression it exerts on the cord.

TREATMENT.—The first and most important element is the treatment of the original cause of the disease, the caries, &c., to which the meningitis is secondary. Any accessible collection of pus should be removed. Rest, counter-irritation to the spine, especially the actual cautery, sedatives to relieve the pain, and tonics in chronic cases are the most important measures. The general treatment for internal meningitis is also suitable to the external form.

INTERNAL MENINGITIS.

Internal meningitis, inflammation beginning within the dura-matral sheath, may be either acute or chronic. The acute form usually commences in the pia mater and arachnoid, the chronic forms may begin in these membranes, or in the inner surface of the dura mater. Little is known practically of any separate affection of the arachnoid.

ACUTE INTERNAL MENINGITIS.

Acute internal meningitis, since it begins usually in the pia mater and arachnoid, has been termed "acute leptomeningitis" but it only remains limited to those membranes when of slight degree. In other cases the inner surface of the dura mater is also inflamed. Frequently the inflammation extends rapidly to the cord, and to such cases the term "meningo-myelitis" is often applied. The inflammation may be either simple or purulent, or it may be secondary to tubercle. It also occurs in conjunction with cerebral meningitis in epidemic form. Acute simple meningitis is a rare disease.

CAUSES.—Little is known of the predisposing causes of acute meningitis, except that the disease is more common in males than in females and in early adult life than at other periods, and that its occurrence is favoured by depressing influences of various kinds, especially by over-exertion. Among traceable exciting causes, the most important is exposure to cold, such as is involved in sitting on damp grass, remaining in wet clothes, exposure to cold winds during perspiration. It may result also from various traumatic influences, from fracture to simple concussion, and from surgical procedures, such as puncture of the sac of a spina bifida. Insolation, prolonged exposure of the back to the heat of the sun, has been the apparent cause in some instances. Suppression of the menses has been regarded as a cause; and perhaps the predisposition to suffer is greater at the menstrual period, but the suppression is more probably a coincident effect of the cause of the meningitis. It has been observed, in rare cases, to come on in the course of acute febrile diseases, scarlet and typhoid fever, acute rheumatism, pneumonia, and in the course of septicæmia. It may also occur by extension, first from the cerebral membranes in cases in which the intracranial inflammation is of local origin, and, secondly, from adjacent disease, outside the spinal membranes. External meningitis is first produced, and therefore all the causes of external meningitis may also be occasional causes of internal meningitis. In septicæmia the inflammation is always purulent. When due to other causes it may be simple, but in most conditions, an acute inflammation may go on to the formation of pus. In some instances no cause can be traced.

Even purulent meningitis occasionally develops from some thus obscure cause, especially in children.

PATHOLOGICAL ANATOMY.—Internal meningitis is usually of wide extent, since the inflammation spreads readily in the loose tissue of the arachnoid. Probably also the movement of the cerebro-spinal fluid aids in the extension, by conveying from one part to another irritant material. In the earliest stage, of which few observations exist, the only change is congestion of the pia mater, which is reddened from vascularity, and dotted with ecchymoses. The inner surface of the dura mater, and the substance of the spinal cord, may be similarly congested. When the inflammation is further advanced, in the stage in which the condition most often comes under observation, the pia mater and arachnoid are opaque and thickened, and an “exudation” of inflammatory products, greyish yellow in tint, may cover the pia mater and occupy the meshes of the arachnoid. The exudation may be semipurulent in aspect, and in the suppurative form the structures are bathed in pus. The inner surface of the dura mater usually presents similar changes, and the inflammatory products may fill up the whole space between the dura and pia mater, thus connecting the two membranes and surrounding the nerve-roots (Fig. 81). The microscope

FIG. 81.



FIG. 82.

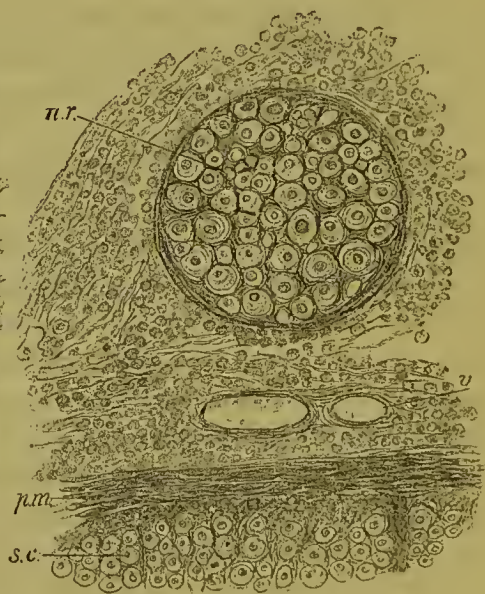


FIG. 81.—Purulent meningitis; portion of spinal cord and membranes; the space between the pia mater and dura mater is occupied by inflammatory products, pus, &c., in which the nerve-roots, *nn*, are embedded. *aa*. Cavities which had apparently been filled with liquid pus. From a case of septic origin, secondary to caries of the jaw.

FIG. 82.—From the same; meshes of arachnoid infiltrated with pus-corpuscles. A nerve-root, *n. r.*, although surrounded by pus, is perfectly normal, and so also are the pia mater, *p. m.*, and the peripheral layer of the spinal cord, *s. c.*

shows abundant leucocytal elements, and the larger round and spindle

cells that are common in all inflammatory products. The former corpuscles, which resemble, and are probably identical with pus-corpuscles, may be abundant, even when the exudation has not a distinctly purulent aspect, and when it has they constitute almost the whole of the material, lying among the fibres of the arachnoid (Fig. 82). The vessels are dilated, and their sheaths distended with cells. The spinal fluid is increased in quantity, and turbid from flocculi, or even purulent in aspect. The nerve-roots are covered with exudation, and are often swollen and reddened from invasion by the inflammation. But they do not always suffer, even in purulent meningitis, as Fig. 82 shows, in which a nerve-root is almost normal in aspect, and its sheath is unaffected, although it is surrounded by pus-cells. The spinal cord is often invaded by the inflammation; it is reddened, pale, and softened, and the microscope shows the tissue changes common in other forms of myelitis. The change is always most marked in the periphery of the cord, and may occur in wedge-shaped areas, having the apex directed inwards, and coalescing at the periphery. The continuity of the vessels, and lymphatic spaces, of the pia mater and cord renders the invasion of the latter intelligible. In some cases of purulent meningitis the pia mater itself is little affected, even when the arachnoid is filled with pus, and in these cases the spinal cord may be normal, as in the example shown in Fig. 82. If recovery takes place, the inflammatory products may undergo cicatricial changes, the membranes remaining opaque and adherent, and there may be permanent excess of arachnoid fluid. The changes in the cord may lead to sclerosis, widely spread, or limited to certain spots, and from these secondary degenerations, ascending and descending, may develop. The area affected varies in different cases. Occasionally the membranes are involved in their whole extent. Usually the affection is greater on the posterior than on the anterior surface; probably on account of the influence of the recumbent posture.

In tubercular inflammation the amount of exudation is usually less, and it may be absent. If present it is commonly grey and gelatinous in appearance, and in it are scattered the greyish or whitish tubercular granulations. Similar granulations may usually be found in abundance upon the inner surface of the dura mater. Such grey granulations are often found upon the spinal membranes in cases of tuberculosis when there is no inflammation and even when the cerebral membranes are intensely inflamed. The arachnoid, especially that covering the cauda equina, may appear as if dusted over with grey particles, so abundant are the granulations.

In many cases, the signs of spinal meningitis are associated with those of inflammation of the membranes of the brain, chiefly of those about the base and posterior part of the brain, rarely of the vertex. The continuity of the spinal and cerebral inflammation may be obvious or indistinct. In the latter case, the connecting inflammation has

apparently been much slighter than that in the base of the brain or around the cord. In cases of slight cerebral meningitis, in which the patient has been recumbent, signs of spinal meningitis, opacity of the arachnoid and its distension with slightly turbid fluid, may exist only in the cervical region, ceasing opposite the upper dorsal vertebræ, and thus extending as far as gravitation (the head being slightly raised on a pillow) favoured the descent of inflammatory products. In the violent purulent cerebro-spinal meningitis which is occasionally produced by a septic influence, pus around the cord is often continuous with that which bathes the base of the brain. In a case of this kind, in which the meningitis was secondary to acute double purulent otitis, Leyden found abundant active micrococci, very similar to those associated with erysipelas.

SYMPTOMS.—Slight pain in the back, and malaise, may precede the acute onset of the symptoms. This is usually marked by a rigor, by pyrexia, and by severe pain in the back. The pain varies in position, according to the locality of the inflammation. It is often felt along the whole extent of the spine. Pain also radiates along the distribution of the nerves, round the trunk or to the extremities. This is paroxysmal and very intense; sharp, darting, burning, or constricting. The pain in the back is usually constant, with exacerbations. It is often increased by movement, usually also by pressure and by the application of a hot sponge to the skin. It is no doubt due to the irritation of the inflamed meninges, while the radiating pain is produced by the inflammation of the sensory nerve-roots. Muscular spasm usually comes on at the same time as the pain. It shows itself first in rigidity of the muscles of the back, most marked, if the inflammation is local, in the neighbourhood of the inflamed part. It may merely cause retraction of the head, or stiffness of the back, or may be so general and severe as to cause opisthotonos, resembling that of tetanus. The spasm usually involves also other trunk-muscles, especially those of the abdomen, which become hard and cramped. The limbs also become rigid, and painful cramp-like spasms occur in them, especially on attempts to move. The spasm is probably in part due to the irritation of the motor nerve-roots, and is in part reflex from the irritation of the sensory fibres distributed to the pia mater. There is usually great hyperæsthesia of the skin to all forms of sensation, and also increased sensitiveness of the muscles, especially in the lower extremities. Pressure on the muscles of the arms may cause no uneasiness, while a similar pressure on those of the legs occasions great pain. Reflex action is usually increased at the beginning. There are constipation and often retention of urine, in spite of irritable attempts to pass water, the result, apparently, of reflex spasm of the bladder and sphincter. The vascular dilatation which follows a scratch on the skin is prolonged (meningeal streak). Dyspnoea may result from the spasm of the thoracic muscles, and may be almost suffocating in its

severity. The pulse may be frequent or retarded. The temperature is raised, sometimes, however, to only a slight degree. Cerebral symptoms, headache, delirium, coma, occur when inflammation has extended within the skull. The "Cheyne-Stokes breathing" may be present, from implication of the medulla.

As the disease progresses, the symptoms of irritation give place to those of paralysis, which may be most marked where the rigidity was greatest. The limbs become relaxed, and feeble or powerless. Sensibility is lessened or lost. Reflex action disappears, and death may occur from asthenia, or from paralysis of the respiratory muscles. Towards the end there is sometimes considerable rise of temperature. In some cases the symptoms become less progressive and the disease passes into a less acute stage; the pains persist, the loss of power continues and may even slowly increase. Death may occur, after weeks of suffering, from the effects of bedsores or from secondary kidney disease, due to the retention of urine, and facilitated by trophic disturbance. On the other hand, in slight cases, the signs of irritation may lessen and pass away, while those of structural damage, paralysis and anæsthesia may remain, and to these may be added muscular atrophy and contractures, from the secondary consequences of the lesions of the nerve-roots. Such persistent symptoms vary much in extent and degree, according to the position and intensity of the morbid process. The symptoms of damage to the cord may slowly increase, in consequence of the spread of chronic myelitis, or still more chronic degeneration, set up by the acute mischief. In very slight cases the symptoms of meningitis may pass entirely away.

The symptoms above described vary in their grouping, according to the position of the disease. When the membranes over the lumbar enlargement are chiefly affected, the pains, hyperæsthesia, and cramps are confined to the legs and loins. When the disease is in the dorsal region there may be similar hyperæsthesia and spasm in the legs, but the pain and cramp extend higher, and involve the trunk. If the cervical region is affected, the symptoms extend to the upper extremities, the dyspnoea may be great, and there is often difficulty in swallowing. Contraction or dilatation of one or both pupils may occur. Extension to the brain is marked by vomiting, general headache, delirium, and paralysis of cranial nerves. If such symptoms preceded those of spinal meningitis, we may conclude that the inflammation commenced within the skull, and descended to the spine. The symptoms vary somewhat according to the nature of the inflammation. In purulent meningitis, strange to say, the symptoms of irritation are sometimes very slight, apparently because there is little tendency to invade the nerve-roots. In the case from which Figs. 81 and 82 were taken there were hardly any symptoms to suggest meningitis, and, although there was paraplegia, it was probably produced by the mere pressure of a large collection of pus on the spinal cord. There were no pains or spasm. The case was apparently of septic origin. The duration of

the acute symptoms varies from a day or two, in severe cases, which end in death, to two or three weeks, in cases of less severity, which may end in either death or recovery. The duration of the subacute and chronic symptoms that supervene is to be measured by months, and sometimes by years.

The symptoms of tubercular spinal meningitis resemble those which have been described, but are usually less intense. There is pain in the back and loins, with stiffness of the spine and retraction of the neck, so that it may be difficult to bend the head forwards. There is also variable rigidity, hyperæsthesia, and tingling in the limbs, followed by lessened sensibility and paraplegic symptoms. Herpes along the course of an intercostal nerve has been noted.

DIAGNOSIS.—The diagnosis of the disease rests on the pain in the back, the rigidity of the spine, the hyperæsthesia and spasm in the limbs, excited especially by attempts to move them, the acute onset of the symptoms and their association with pyrexia. In simple *myelitis*, on the other hand, there is little pain in the back, paralysis occurs early and is the characteristic symptom, and there is little or no spasm in the limbs in the early stage of the affection. Often, however, some meningitis occurs at the onset of acute myelitis and then some pain in the back and slight rigidity in the limbs may precede or accompany the onset of the paralysis. In such cases the predominance of the meningeal or cord symptoms must determine the category in which the case is to be placed. *Tetanus* is attended by rigidity of the back, and by spasm, but there is no fever at the onset, trismus is an early and obtrusive symptom, and the paroxysms of muscular spasm are excited by peripheral impressions much more readily than in meningitis, in which they occur chiefly on attempts to move. *Rheumatism of the dorsal muscles* may cause pain in the back on movement, but there is not the spontaneous pain, or the spasm in the muscles, which characterise meningitis. In *gonorrhœal rheumatism* both spinal and radiating pain may exist, probably from an affection of the vertebral articulations, similar to that which exists elsewhere. The affection of other joints and the absence of rigidity are commonly sufficient for the diagnosis. The diagnosis of tubercular spinal meningitis depends on the combination with cerebral meningitis, which usually precedes the spinal symptoms, and on the gradual and insidious onset. Indications of the tubercular or scrofulous diathesis are commonly present in the state of other organs, or to be ascertained from the family history of the patient.

PROGNOSIS.—The prognosis is grave in all cases. It is worse the more severe and acute are the symptoms, the higher the temperature, and the sooner the symptoms of irritation give place to those of paralysis. It is worse also when due to lesions of the spinal column or to tuberculosis, than when due to cold, and worse in the so-called "spontaneous" cases than in those which result from traumatic causes.

Recovery is more probable in middle life than in early or advanced age. The previous health of the patient also influences the prognosis. It must always be remembered that even if the patient survives the period of acute inflammation, serious permanent mischief may remain.

TREATMENT.—Perfect rest and quiet are of the greatest importance throughout the course of the disease. All noise should be, as far as possible, excluded; the light should be subdued, and all bodily movement and mental exertion as far as possible avoided. Although it is undesirable that the spine should be the lowest part of the body, yet, in acute meningitis, it is scarcely possible for the patient to lie in any other posture. The prone position interferes with respiration, and both it and a lateral posture cannot be borne on account of the greater muscular exertion which, directly or indirectly, they entail. Dry, or, in robust patients, wet cupping, or leeching, along the spine may be employed at the onset, and be followed by the local application of ice, by means of a spinal ice bag which may be extemporised with gutta-percha tissue made to adhere by touching it with chloroform. Counter-irritation, by blisters or repeated sinapisms, is more useful when the disease is subsiding than at the onset. In cases that are due to cold, free diaphoresis often does good; a hot air or vapour bath should be employed at the onset of the treatment. A warm bath may be followed by moist packing for several hours. The relief thus given is sometimes very great. The bowels should also be freely opened.

The only internal remedy which has been held in general repute, as capable of influencing the inflammatory process, is mercury. The confidence placed in it of old is not altogether unwarranted. It may be given, guarded with opium to avoid irritation of the bowels, until there is a very gentle affection of the gums. The oleate of mercury may be rubbed in along the spine. It irritates the skin and then may combine some counter-irritant influence. Iodide of potassium seems to have little influence over acute inflammation.

It is necessary to give sedatives for the relief of the pain and spasm. Of these morphia, given by the skin or the mouth, is alone really effective. Sometimes inhalations of chloroform are necessary to relieve the intense suffering. In slight cases relief may be afforded by belladonna, or by atropin injected beneath the skin. Choral, or chloral and bromide, may be given if there is insomnia.

When the disease has passed into a chronic stage, iodides have been thought useful. Counter-irritation may be freely employed with advantage. Tonics, iron, quinine, and even strychnia are beneficial. Warm baths, as those of Bath and Aix les Bains, seem sometimes of service, especially employed as hot douches to the spine. The local consequences, muscular atrophy, contracture, &c., need special local treatment by electricity, rubbing, and the like.

Epidemic cerebro-spinal meningitis is described in the section on diseases of the cerebral membranes.

CHRONIC INTERNAL MENINGITIS

Chronic inflammation of the membranes of the cord, within the dural sheath, is divided into two forms, according as it begins in, and chiefly affects, the dura mater, *chronic internal pachymeningitis*, or the pia mater and arachnoid, *chronic leptomeningitis*. Although these forms sometimes present distinct clinical and pathological features, they have, when of considerable degree, many characters in common; they own the same causes, and need the same treatment. Hence it is most convenient to describe them together as forms of internal meningitis. The condition termed *hæmatoma of the dura mater* depends on a form of hæmorrhagic inflammation.

CAUSES.—Chronic internal meningitis, in every form, is most frequent in adult age, and, like acute inflammation, affects men more frequently than women. It has been observed in persons with neurotic heredity, although whether any influence is to be ascribed to this relation is doubtful. Debilitating influences of various kinds may predispose to the disease, and it is sometimes the result of constitutional syphilis and of chronic alcoholism. The syphilitic form often reaches a high degree of intensity. Among exciting causes the most important is prolonged and habitual exposure to cold. It is doubtful whether simple mechanical congestion ever produces the disease. Traumatic lesions, concussion, &c., are occasional causes, and the affection has been ascribed to prolonged over-exertion and to sexual excess. It may result by extension from inflammation outside the dura mater and from chronic inflammation of the substance of the cord itself. Chronic cerebral meningitis is sometimes associated with the spinal inflammation, but it is often uncertain whether the association is due to extension or whether both are the result of a common cause. Lastly, it must be noted that chronic meningitis may be a sequel to the acute form. Hæmorrhagic pachymeningitis occurs especially in the insane, but has been met with as a consequence of chronic alcoholism and after injuries.

PATHOLOGICAL ANATOMY.—In slight and moderate degree there is merely opacity and thickening of the membranes affected, sometimes with distension of vessels or minute spots of extravasation. The inner surface of the dura mater may be granular. The spinal fluid is increased in quantity and is turbid. The opacity of the arachnoid may be such that the spinal cord cannot be seen through it. When the changes are greater in degree, the dura mater and pia mater may be connected together by a tract of inflammatory tissue of considerable thickness, so that it may be impossible to say in which membrane the disease commenced. The microscope shows the ordinary elements of inflammatory tissue, cells of various kinds, many lymphoid and pus-

like corpuscles, and distended vessels, often encrusted by lymphoid cells. Frequently also the pia mater is transformed into a thick irregular layer of homogeneous tissue in which no distinct cell-elements can be perceived, and only faint indications of a fibrous structure (Fig. 83). The walls of its vessels may be greatly thickened by similar material.

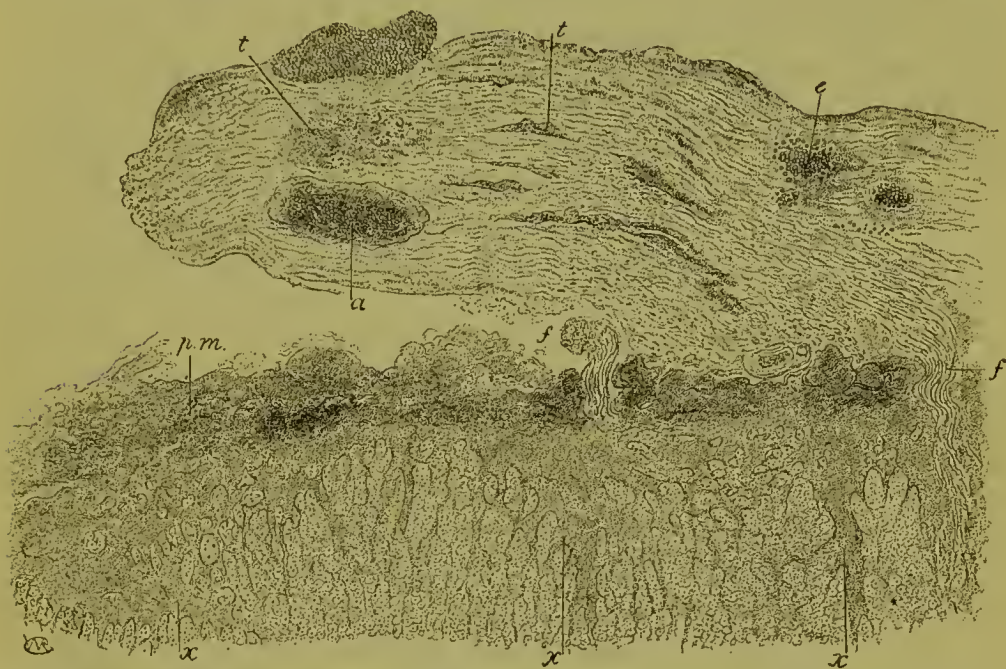


FIG. 83.—Chronic alcoholic meningitis. Section of edge of anterior column and of a large nerve-root; carmine preparation. *p. m.* Pia mater irregularly thickened and transformed into amorphous-looking tissue, from which wedge-shaped branching tracts (*x x*) extend into the white substance; *f. f.*, fasciculi of nerve-fibres entering the cord; *a.*, an artery in the nerve-root, enlarged and with thickened walls; *t. t.*, tracts of amorphous connective tissue; *e.*, a small extravasation.

The nerve-roots passing through the diseased membranes are usually inflamed, reddened, and swollen, and afterwards may be compressed and atrophied, if the amount of new tissue formed about them is considerable. They suffer thus especially when the inflammation involves the dura mater, on account of the unyielding character of the fibrous sheaths which they receive from that membrane. When the pia mater is thickened, scattered tracts of connective tissue may be seen in the substance of the nerve-roots, between the fibres (Fig. 83, *t*).

The spinal cord may be little affected, but is frequently damaged by the extension to it of the inflammation, causing softening, vascularity, loss of distinction between grey and white substance, breaking down of nerve-elements, and infiltration by lymphoid and angular cells. It may also suffer, when the amount of newly-formed tissue is considerable, by compression alone or in combination with the inflammation which usually accompanies pressure. When the pia mater is thickened, tracts of similar tissue may extend into the cord, becoming

narrower as they pass inwards and sending out branching trabeculæ (Fig. 83, x). Between these tracts the nerve-fibres may be normal or degenerated.

The membrane in which the inflammation begins may be much more affected than the other, although the latter is very rarely healthy except in the slightest cases. Inflammation beginning in the pia mater and arachnoid is often extensive in range. That which commences on the inner surface of the dura mater is frequently localised. This local inflammation of the inner surface of the dura mater often leads to the formation of a large amount of new tissue, and has been described by Charcot and Joffroy as *hypertrophic internal pachymeningitis*. It affects most commonly the cervical region, but sometimes occurs at the lower part of the cord. On opening the spinal canal a fusiform tumour is seen, the outer surface of which is the unaltered outer surface of the dura mater, and on section the enlargement is seen to depend chiefly on a great thickening of the inner part of this membrane, sometimes amounting to one fifth of an inch; several layers of new tissue can often be distinguished. The pia mater may be normal, but is more commonly thickened, and it is often united to the tissue proceeding from the dura mater. The cord is compressed and commonly softened at the spot, and it presents signs of old inflammation. The nerve-roots are also greatly damaged by compression by the newly-formed tissue. In other cases the thickening of the dura mater is slighter and more diffuse, affecting occasionally a wide extent of the membrane. Sometimes at the spot most affected, the cord may be surrounded by a ring of new tissue of cartilaginous hardness. The vertical extent of the disease varies much. It is often considerable, sometimes universal, but occasionally it is small. The membranes about the cauda equina may alone be affected—the dura and pia mater adherent, and the nerves united in a fibrous mass.

It is doubtful whether the white fibroid or cartilaginous plates found so often in the arachnoid after death have any connection with preceding inflammation. In most cases in which they are found no symptoms have existed during life. It is said, however, that when they are numerous and extensive they may give rise to symptoms closely resembling those of chronic meningitis. (Vulpian). Fibroid plates in the dura mater have been seen in a case in which previous symptoms suggested that they resulted from chronic inflammation. (Jaccoud.)

In the rare cases in which the inner surface of the dura mater is the seat of a form of hæmorrhagic inflammation similar to, and commonly associated with, that which affects the cranial membrane (internal hæmorrhagic pachymeningitis, hæmatoma of the spinal dura mater), a reddish brown exudation covers the surface of the membrane. It is composed of fibrin and extravasated blood; the latter may be encysted in small cavities, or may be in various stages of transformation. The change commonly extends over a great part of the cord. It is apparently the result of hæmorrhage into inflammatory tissue.

SYMPTOMS.—The symptoms of chronic internal meningitis, like those of the acute form, are due to the irritation of the membranes, to the damage to the nerve-roots, and to the affection of the cord. In the different forms, one or another of these sets of symptoms predominate, so that the varieties, while possessing many characters in common, may differ considerably in their clinical aspect. In some cases, we can infer, with accuracy, the special form of the disease, but in many others we cannot carry our diagnosis beyond the conclusion that chronic internal meningitis exists, and are unable to say, even with probability, which membrane is primarily affected.

The common symptoms are these: There is pain in the back, increased by movement, sometimes, however, amounting only to heavy dull discomfort, and accompanied by some stiffness of the back, and, when in the cervical region, by some retraction of the head. The pain is increased by pressure on the spines, and its increase by movement may cause a fixation of mobile parts, as the neck, in some abnormal position. The pain is apparently due to the irritation of the meningeal nerves, and the rigidity is to be regarded as a reflex effect of this irritation.

Still more conspicuous, in many cases, are the radiating or excentric pains due to the irritation of the nerve-roots, and referred to the region in which the nerves are distributed, back of the head, neck, arms, thorax, abdomen, loins, or legs, according to the position of the disease. These pains are often very severe, sharp, darting, burning, or rheumatoid in character, paroxysmal in occurrence, and sometimes worse at night. They may be accompanied by a painful sense of constriction, and by various unpleasant sensations, numbness, tingling, formication, in the same areas. Hyperæsthesia may also exist, so that pain, sometimes thrilling in character, is produced by touching the skin, or by slight degrees of heat or cold, and a hot or cold sponge passed along the skin of the back may reveal the chief seat of disease by the change of sensation at its level. The hyperæsthesia may be more marked to one form of sensation than to another. Cutaneous eruptions, such as result from other nerve-lesions, have also been observed in association with the pains. In the same regions, muscular twitching, tremor, or even spasm may result from the irritation of the motor root-fibres. These symptoms correspond in their level to the part of the membranes chiefly diseased. When the lumbar membranes are affected, the radiating pains are felt in the legs, but when the disease is higher up, the legs may merely feel heavy, and be the seat of slight abnormal sensations.

These pains having lasted for a time, weeks or months, certain paralytic symptoms manifest themselves. The pains sometimes persist, but may cease when paralysis comes on. Some paralytic symptoms occur in the regions in which the severe pains are felt, and depend on further damage to the nerve-roots. Sensation becomes lessened, or even lost, in certain areas, to touch, or pain, or both. The muscles

in the same region become weak, and waste; their irritability to faradism is lessened, that to voltaism may persist or may also become less than normal. The wasting affects the muscles irregularly, but entire groups may atrophy. Reflex action in these parts is lowered. In the trunk this loss of reflex action, and anæsthesia, may be the chief local symptoms. As the disease progresses, the cord itself suffers, by compression, or by extension to it of the inflammation. The parts below the seat of the disease then become paralysed, the legs are weak and the seat of dull heavy pain; reflex action in them may be increased, while their muscular nutrition continues good, unless the lumbar membranes are diseased, or the myelitis descends the cord. Power over the sphincters may be lost and bed-sores may form.

When the inflammation affects chiefly the pia mater and arachnoid (*chronic leptomeningitis*) the symptoms of meningeal irritation are very conspicuous,—vertebral pain and rigidity of the back, with cutaneous hyperæsthesia, and the radiating pains may also be considerable. These symptoms are especially marked in cases of subchronic meningitis, such as are due to chronic alcoholism. The local paralytic symptoms, anæsthesia and muscular wasting, may be absent in these cases, or may only come on in slight degree after the other symptoms have lasted for a considerable time. On the other hand, weakness and pains in the legs, from affection of the cord itself, may be an early symptom.

When the inflammation begins in the dura mater (*internal pachymeningitis*) the symptoms of spinal irritation, pain and stiffness of the back, although occasionally present, are much less prominent symptoms than are those which depend on the irritation of, and damage to, the nerve-roots. The radiating pains are very severe and are often the earliest symptom. At a later stage the muscular weakness, followed or accompanied by wasting, chiefly attracts attention, and the wasting may be so great that the disease is mistaken for progressive muscular atrophy. This is especially the case when the lesion is in the cervical region, the “cervical hypertrophic pachymeningitis” of Charcot and Joffroy. In this affection, pains in the back of the head, neck, shoulders, and arms, often accompanied with a painful sense of constriction, precede muscular atrophy. The wasting may involve many muscles, but usually those supplied by the radial nerve are much less affected than the others; the escape of the long extensors of the wrist and fingers, while the flexors of the wrist and fingers and the interossei atrophy, leads to a peculiar deformity; the unopposed muscles cause persistent over-extension of the wrist; the phalangeal joints are flexed and the metacarpo-phalangeal joints are extended, but from the position of the wrist they are not over-extended, and the hand thus differs from the claw-like hand (p. 30). With this muscular wasting in the arms, there is paraplegic weakness, from the pressure on the cord. In rare cases the disease affects the membranes over the lumbar enlargement or cauda equina, and then the legs are the seat of the pains,

paralysis, and atrophy, already described, and the sphincters are early paralysed.

The pachymeningitis may be confined to the dorsal region. In such a case cutaneous anæsthesia over the whole region supplied by the dorsal nerves, was associated with paraplegia from compression of the cord. In another instance areas of anæsthesia, with severe pains, existed over the legs, trunk, neck, and back of the head, without any indication of damage to the cord itself.

The symptoms of syphilitic meningitis do not differ from those just described and may present the characters of an affection beginning in either the pia mater or dura mater. As already stated, the pain and muscular wasting often reach a high degree in these cases.

In hæmorrhagic pachymeningitis the symptoms are those of a slight chronic meningitis, pain in the back or extremities, interference with movement, weakness of muscles, and anæsthesia of the skin. It has never been diagnosed during life.

DIAGNOSIS.—The diseases with which chronic internal meningitis is liable to be confounded, are different according as the symptoms of irritation, of damage to the cord, or of compression of the nerve-roots, predominate.

The spinal pain and stiffness may be mistaken for simple rachialgia or “spinal irritation,” but in this disease the whole spine is tender, several separate points are much more tender than the rest, and there are not the radiating pains or paralytic symptoms of meningitis.

Peripheral pains, especially about the trunk and legs and shoulders, are prominent symptoms in some cases of muscular rheumatism, or rheumatic neuralgia. In these, however, there is not the spinal pain and tenderness, and although there may be apparent increased sensitiveness in the region of the pains, it is of the deeper structures and not of the skin.

The muscular wasting may be mistaken for atrophy due to degeneration or subacute inflammation of the anterior cornua of the cord (progressive muscular atrophy, subacute spinal atrophy). The cervical “hypertrophic” form of meningitis bears special resemblance to this disease, since the wasting in the arms is often associated with paraplegic symptoms in the legs. The distinction is that the atrophy, in the meningeal form, is preceded by severe pains, and often accompanied by areas of hyperæsthesia or anæsthesia, and is random in distribution. In chronic affections of the anterior cornua both pains and anæsthesia are usually absent and the wasting often affects the muscles in a certain order, which will be described in the account of the disease. Thus in a man who was supposed to have cornual disease on account of wasting of some muscles of both legs, irregularly distributed, a history of severe sharp pains, and the discovery of a patch of anæsthesia on one leg, and the presence of a burning pain round one side of the trunk, and the irregular distribution of the wasting, led to a diagnosis of

chronic meningitis damaging the nerve-roots. There was a history of syphilis, and appropriate treatment completely removed the symptoms. Chronic disseminated myelitis may cause muscular wasting, but does not cause the pain in the back, or the rigidity of the spine, which occur in meningitis. The paralysis is usually out of all proportion to the wasting and anæsthesia. It must be remembered that the two conditions of meningitis and myelitis are often combined, and the fact has to be recognised in our diagnosis.

When the meningitis is lumbar, the pains in the legs may resemble those of locomotor ataxy, but the absence of rigidity in tabes and the presence of ataxy, distinguish it from meningitis. The knee-jerk may be lost in each disease, but in meningitis there is muscular weakness and wasting, often with altered electrical reaction, symptoms never present in the early stage of tabes. It may be noted, however, that there is sometimes a thickening of the pia mater over the posterior columns when these are sclerosed. This thickening is due to a process partaking of the nature of inflammation. It may account for the vertebral pain occasionally felt in that disease, but does not cause any other symptom of meningitis. The greatest resemblance to chronic meningitis is presented by some cases of tabes in which the cord disease reaches a greater degree in the dorsal than in the lumbar region. There are then pains and anæsthesia in the trunk, and the atrophy in the legs may be little marked. In meningitis, if the membranes over the lumbar enlargement are normal, the knee-jerk is preserved and often increased, while it is lost in tabes. The anæsthesia is more general and uniform in tabes than in meningitis.

When chronic meningitis affects the cervical region it may be mistaken for the tonic form of torticollis, but may readily be distinguished by observing, first that the head is not fixed by spasm, since the muscles in a state of tension are not those by which the posture of the head would be produced, and secondly that the fixation is distinctly due to the pain which movement occasions.

Caries of the spinal bones and chronic meningitis have many symptoms in common, and necessarily, since chronic local meningitis is one of the effects of the bone disease, and produces many of the symptoms by which the latter is recognised. The diagnosis can only be made by the detection of the signs of bone disease enumerated on p. 170.

PROGNOSIS.—In all forms and degrees of chronic meningitis, except the most trifling, the prognosis is serious. The severe degrees of the affection are attended with danger to life, and the slighter forms entail serious consequences, since many effects of the disease, especially the damage to the spinal cord itself, tend to increase by the degenerative tendency which is set up. The neuralgic pains, which result from the damage to nerve-roots, are extremely obstinate. But in many cases the symptoms pass away under appropriate treatment.

The prognosis is most favorable in the cases which result from concussion of the spine and from syphilis. But in all cases, even those of syphilitic origin, in which there is reason to infer considerable formation of new tissue, the prognosis must be cautious, since the cicatricial contraction of this tissue may perpetuate the damage to cord and nerves, which its pressure has produced. The prognosis is better, in other cases, in proportion to the general health of the patient, to the moderate degree of the mischief, and to the extent to which its causes are under control.

TREATMENT.—Rest is essential. In some cases it should be absolute, and in all cases movements which increase pain should be avoided. The pain is the expression of mechanical irritation, which hinders recovery. The prone couch, impracticable in acute meningitis, can often be employed in chronic cases with advantage, to lessen mechanical congestion of the spine. Warm baths and diaphoretic baths have been recommended. They are most useful in subacute cases. Next in importance to rest is counter-irritation. Repeated sinapisms or stimulating liniments may be used if mild counter-irritation only is required, but in most cases greater good will result from more energetic means, blisters, the thermic hammer, or the actual cautery. The latter is especially recommended by Joffroy for the hypertrophic pachymeningitis.

Sedatives are usually needed to relieve the pain; injections of morphia, chloral, Indian hemp, or sedative liniments of chloroform, belladonna, &c. In employing sedatives it must be remembered that they will usually be needed, in chronic cases, for many months, and after a time, by the artificial need they create, they may apparently keep up the pain. It is therefore well, after they have been used for some time, to change the sedative. If injections of morphia have been employed for a considerable time, it is well to see the effect of an occasional injection of plain water.

Iodide of potassium and mercury are the agents that have most influence over the morbid process itself. Both are effective in syphilitic cases. In other forms of the disease mercury has far more influence than iodide. In many cases tonics, iron, quinine, and cod-liver oil are needed. The muscular wasting which results from damage to the nerve-roots requires treatment by galvanism, rubbing, and by passive movement to prevent contractures. The treatment of hæmorrhagic pachymeningitis is the same as that of the ordinary form, but if it be suspected, injections of ergotine should be tried. Treatment appears, however, to exert little influence upon it.

HÆMORRHAGE INTO THE SPINAL MEMBRANES; SPINAL MENINGEAL HÆMORRHAGE; HÆMATO- RACHIS.

Blood may be extravasated outside the dura mater, between it and the bones (extra-meningeal hæmorrhage), or within the dura mater (intra-meningeal hæmorrhage). In the latter situation the blood may be in the subdural space, between the dura mater and arachnoid (subdural hæmorrhage), or it may be beneath the arachnoid, between it and the pia mater (subarachnoid hæmorrhage). Extravasations outside the dura mater are more common than those within it.

CAUSES.—Meningeal hæmorrhage may occur at all ages. It is met with in newly-born children, but in them is probably of traumatic origin. Spontaneous hæmorrhage is unknown in early childhood, but occurs at all other ages, and is more common in men than in women. No relation has been traced between its occurrence, and the general conditions of circulation, increased blood-pressure, vascular degeneration, &c., which favour the occurrence of cerebral hæmorrhage. It is doubtful whether it is predisposed to by chronic alcoholism. Of the immediate causes, injury is the most frequent, fractures of the spinal column, blows or falls on the back which do not fracture the vertebræ, and even falls on the feet or buttocks. It is occasionally found after death from severe convulsions, epilepsy, puerperal eclampsia (in which an altered state of the blood may assist), chorea, strychnine poisoning, and tetanus. In these cases the blood may be outside or within the dura mater. In many instances no symptoms were observed during life which could be referred to the hæmorrhage, and it is probable that the extravasation occurred only during the last moments of life. In some cases, however, the muscular spasm, resulting from the meningeal hæmorrhage, may have been mistaken for an independent symptom, and the hæmorrhage, which was really the cause of the spasm, regarded as its consequence. It has probably been so in some of the cases of the so-called tetanus of newly-born children. In two-thirds of these cases extra-meningeal hæmorrhage is found, and in those in which the tetanoid symptoms occur almost immediately after birth, they are probably due to the extravasation. Severe and prolonged muscular exertion has been, in a few cases, the apparent cause of spontaneous hæmorrhage. It also occurs in some diseases in which there is a hæmorrhagic tendency, such as purpuric states, and the hæmorrhagic forms of some acute specific diseases, smallpox, yellow fever, &c., and very rarely in typhoid fever, apart from any hæmorrhage elsewhere. In most of these cases its occurrence has not been suspected until the post-

mortem examination revealed it, and it probably occurs generally during the last moments of life. A very rare cause is the bursting of an aortic aneurism into the spinal canal, after erosion of the bodies of the vertebræ, the blood being effused outside the dura mater. Hæmorrhage within the dura mater has resulted from the rupture of an aneurism of a vertebral artery. Blood may also descend into the spinal membranes from the cranium, in cases in which a cerebral extravasation, especially about the pons, escapes into the meninges. Lastly, in cases of intense meningeal inflammation, ecchymoses, and sometimes considerable extravasations have been found, on both sides of the dura mater or in the pia mater. Thus considerable extra-meningeal hæmorrhage has been met with in cerebro-spinal meningitis.

PATHOLOGICAL ANATOMY.—*Extra-meningeal hæmorrhage* probably comes from the plexus of large veins which lie between the dura mater and the bone. It is usually not of large extent, and the blood accumulates chiefly on the posterior aspect, where, in the recumbent posture, gravitation favours accumulation, and where the space between the membrane and the bone is greater than in front. Sometimes the extravasation is very extensive, covering a large part of the dura mater, and in such cases it may extend through the intervertebral foramina, along the nerves. Hæmorrhage is more common in the cervical region than elsewhere, but may occur in any part. The blood is usually coagulated, wholly or partially. The dura mater is reddened, and sometimes the staining extends through to the inner membranes. If the hæmorrhage is considerable the spinal cord may be compressed, but the amount of blood is not often sufficient for this. When a body has been lying on the back, the veins outside the dura mater become distended with blood, which escapes when they are divided in opening the spinal canal. Care must be taken to avoid the error, on the one hand, of regarding the blood thus escaping as an ante-mortem extravasation, and, on the other, of overlooking a hæmorrhage which actually exists. In all cases of acute spinal disease the body should, if possible, lie face downwards until the examination has been made. In rare cases hæmorrhage has been found within the substance of the dura mater.

Intra-meningeal hæmorrhage.—An extravasation into the sac of the dura mater (subdural hæmorrhage) may be small in quantity, or may fill the whole cavity. When small it may pass from one part to another. In subarachnoid hæmorrhage the blood comes usually from the vessels of the pia mater. It may surround the cord for an inch or two or may fill the whole subarachnoid cavity from one end of the cord to the other. Such extensive effusions are rare, except when the blood descends into the arachnoid from the cerebral membranes. Blood effused into the spinal arachnoid has been known to ascend as high as the pons and even break through the valve of Vieussens and get

into the cerebral ventricles.* When cerebral and spinal hæmorrhage coexist, it must not be at once assumed that the blood has passed from one to the other, since hæmorrhage may occur in each at the same time, as in one recorded case of puerperal eclampsia. (Charrier.)

In meningeal hæmorrhage, cerebral as well as spinal, the spinal fluid is often blood-stained, and thus spinal hæmorrhage may be thought to be more considerable than it really is. The spinal cord is often discoloured and compressed and is especially damaged when the hæmorrhage is beneath the arachnoid. In extensive extra-meningeal hæmorrhage the subarachnoid fluid may be driven away from the compressed part, and may distend the arachnoid beyond the area of compression, the limit of which is thus marked. In cases which have lasted more than a few days there are usually signs of meningitis, set up by the irritation of the blood.

SYMPTOMS.—As already stated, slight meningeal hæmorrhage is sometimes found post mortem in death from convulsive diseases when no symptoms of it were observed during life, and in these cases it is probable that the extravasation occurs during the lethal convulsion. In the cases in which hæmorrhage causes symptoms, these are, for the most part, those of meningeal irritation, and thus bear some resemblance to the symptoms of meningitis. They differ in the suddenness and violence of their onset, to which exceptions are extremely rare. These symptoms are nearly the same whether the hæmorrhage is outside or inside the dura mater.

The first indication of the lesion is usually sudden and violent pain in the back, corresponding in position to the seat of the hæmorrhage, but sometimes felt along a considerable extent of the back, and often severe in the loins. This pain in the back is usually accompanied by pain along the course of the nerves passing through the membrane near the extravasation, darting or burning pains, often of great intensity, making the patient shriek with agony. They are paroxysmal in character, and between the pains there may be abnormal sensations, tingling, &c., and hyperæsthesia, referred to the same parts. Muscular spasm usually coincides with the pain and involves partly the vertebral muscles, causing rigidity of the spine or actual opisthotonos, partly the muscles supplied by the nerves in which the pain is felt, partly the muscles supplied from the cord below the seat of the hæmorrhage. The convulsive movements are sometimes general. Intense pain in the back and general convulsion have been known to be the only symptoms. Occasionally there is persistent contraction of muscles, and there is usually spasmodic retention of urine. These symptoms of irritation are no doubt due in part to the irritation of the membranes (causing the vertebral pain and reflex spasm), and partly to the irritation of the nerve-roots, motor and sensory. Paralytic symptoms quickly follow—

* As in an apparently conclusive case reported by Leprestre, 'Arch. Gén. de Méd.,' xxii, p. 331.

weakness and lessened sensitiveness in the limbs below the lesion. Ultimately there may be complete loss of motion and sensation in the lower limbs, but such absolute paralysis is not common. Loss of power at the onset of the symptoms usually indicates simultaneous hæmorrhage in the cord or the effusion of a very large amount of blood. It occurs, for instance, when an aneurism bursts into the spinal canal.

The symptoms differ in their distribution according to the seat of the disease. When hæmorrhage is in the cervical region (a frequent seat), the pains are felt in the neck and arms, and the rigidity may cause absolute immobility of the neck, while dysphagia, interference with respiration, and dilatation of the pupils may be added to the other paralytic symptoms. When it is in the dorsal region, intense pain encircles the chest or abdomen. If in the lumbar region, the pain is felt in the legs, and there are early paraplegic symptoms, with loss of reflex action in the legs, and paralytic incontinence of urine and fæces.

As a rule, in spinal hæmorrhage, the cerebral functions are unaffected; the unfortunate patient is conscious of all his sufferings from first to last. Occasionally, however, consciousness is lost for a short time, apparently from shock, and even delirium or coma may come on, either as an indirect effect of the spinal lesion on the brain, or in consequence of a sudden increase in the intracranial pressure, due to the displacement upwards of the spinal fluid by the effusion of blood. But in most cases in which cerebral symptoms coexist with those of spinal hæmorrhage, the former have been due to simultaneous intracranial disease. So, in some cases of spinal hæmorrhage in newly born children, apoplectiform symptoms have been present from simultaneous hæmorrhage into the membranes of the brain. In very rare cases the symptoms of spinal hæmorrhage have come on insidiously, without pain, as in a case in which extensive hæmorrhage outside the dura mater in the cervical and upper dorsal region, in a girl of fourteen, caused only very gradual weakness in the arms, and difficulty of breathing, from which she died at the end of a fortnight.*

The symptoms in acute cases usually reach their height in two or three hours, sometimes not for a few days. Death may occur when the symptoms are fully developed, or, after reaching their height, they may decline, to be increased, a day or two later, by secondary meningitis. This is accompanied by some pyrexia and lasts a week or ten days, and then, in favorable cases, permanent improvement commences. It is, however, very common for death to take place when the symptoms have existed a few hours only. It may be due to exhaustion, in consequence of the violence of the pain and spasm, but is more often produced by interference with respiration.

DIAGNOSIS.—In cases in which marked symptoms are produced, the diagnosis rests on the combination of sudden localised pain in the back, with the other evidence of irritation of the membranes, nerve-

* R. Jackson, 'Lancet,' 1869, p. 5.

roots, and cord, above enumerated. When the hæmorrhage is of traumatic origin, the fact that the symptoms rapidly followed an injury facilitates the diagnosis. In the extremely rare cases in which the symptoms come on insidiously, without pain, the diagnosis of the exact nature of the disease is scarcely possible.

In hæmorrhage into the substance of the cord, vertebral pain is much more frequently absent than in meningeal hæmorrhage. The symptoms of injury to the cord itself are prominent from the commencement; sudden paralysis, it may be at first partial, and rapidly increasing, without initial pain. In meningeal hæmorrhage, pain and symptoms of irritation usually precede any considerable paralysis. In cases which recover, the paralytic symptoms are more persistent when the hæmorrhage is into the substance of the cord, than when it is into the meninges. Extravasations into the substance of the cord often break through into the membranes, and the symptoms of both lesions are then combined.

Meningitis is distinguished by the more gradual onset of the symptoms and the presence of fever from the first. It must not be forgotten that meningitis may result from hæmorrhage. In myelitis pain is commonly absent, and the symptoms of irritation are far less pronounced. The diagnosis from tetanus depends on the absence of trismus, and on the presence of severe spinal pains, and on the more sudden onset. Most cases of tetanus come on more or less insidiously. It is chiefly in the case of newly-born children that the diagnostic difficulty will arise. Tetanus is in them most common two or three days after birth; the tetanoid symptoms which may supervene an hour or two after birth are probably not true tetanus but tetanoid spasm, the result of meningeal hæmorrhage produced by injury during birth. One case has been recorded in which the symptoms most closely simulated those of strychnine poisoning. Violent paroxysms of muscular spasm, with intense general pain, but without spinal pain, followed by death in two hours, were apparently due to an extensive hæmorrhage into the sac of the dura mater.* Analysis revealed no strychnia in the stomach. In such a case the diagnosis would have to be guided as much by circumstantial evidence as by the symptoms.

PROGNOSIS.—In all severe cases the prognosis is most grave. A large number of such cases prove fatal in a few hours. If the symptoms reach their height, and cease to increase, and the indications of damage to the cord itself, are moderate, the patient will probably recover, although there is still some danger until the period of secondary inflammation is over. Paralytic symptoms often persist for some time, but even these, in the end, may pass away. At the onset, the prognosis must be governed by the rapidity with which the symptoms develop and by the seat of the disease. Hæmorrhage in the cervical region is more serious than in the lower parts of the cord.

* Dixon, 'Lancet,' 1879, p. 333.

TREATMENT.—The first point to secure is absolute rest. Even passive movement should be avoided as far as possible. Posture is of the first importance. It should be on the face or side, and not on the back, so that the spine may not be the lowest part. Venesection has been employed in strong individuals, with the object of arresting hæmorrhage by rapidly lowering the blood-pressure, and it is a justifiable proceeding if the symptoms are making rapid progress, and the diagnosis is sure. Leeches or wet cupping to the spine, or leeches to the anus, have also been recommended in cases in which venesection is unadvisable. Ice should be applied to the spine in all cases, and ergotine may be injected under the skin or a full dose of ergot given by the mouth. The bowels should be freely opened. Sedatives are usually required to relieve the pain. The stage of meningitis must be treated on the principles laid down for the management of the acute form of that disease. The residual palsy requires treatment by electricity, &c., in the same manner as the consequences of meningitis.

DISEASES OF THE SPINAL CORD.

ANÆMIA AND HYPERÆMIA OF THE SPINAL CORD.

The condition of the vessels of the spinal cord after death, their fulness or emptiness, affords no indication whatever of their state during life. Inferences as to the ante-mortem state, drawn from the post-mortem condition, are altogether erroneous, with the rare exception of local change, in which the vascular condition of one part differs from the rest. Practically, such local variation occurs only in the local hyperæmia that attends inflammation, and the anæmia that results from pressure. Hence the occurrence of variations in the state of the vessels of the cord, and the effects that such variations may produce, are matters of inference from symptoms observed during life, symptoms that are, in themselves, open to various interpretations. Where the ground is barren of facts theory is always luxuriant. Anæmia or congestion of the cord affords a ready explanation of symptoms the cause of which is unknown, and it is scarcely surprising, therefore, that such an explanation has been often given. Some surprise may, however, reasonably be felt at the absolute confidence and precision of detail with which these states have been invoked as morbid processes, when the opinions expressed rest upon not one tittle of definite evidence. Positive assertions always receive some credence, however unwarranted the assertions may be, and positions incapable of proof are sometimes also incapable of disproof. It would be a futile and useless task to

attempt to refute in detail the various statements that have been made regarding the influence of anæmia and hyperæmia of the spinal cord. It will be sufficient to point out briefly what may be reasonably surmised regarding these morbid states.

ANÆMIA OF THE CORD.

Nothing is known of persistent anæmia of the cord from organic obstruction in the arteries lessening the blood-supply. Transient anæmia, if it occurs, must be due to arterial spasm, dependent on the vaso-motor nerves. This has been assumed as an explanation of certain transient symptoms occasionally observed, loss of power in the legs, and sensory disturbance, tingling, "pins and needles," &c., in the legs and arms, and the tetanoid cramp and tingling in the hands with which patients sometimes wake up. The theory is tenable, but it is also conceivable that such symptoms are due to transient functional states of the nerve-cells of the cord. A functional derangement of these cells must be the immediate cause of the symptoms, whether such derangement is primary or is produced by spasm of the vessels.

In general anæmia, such as occurs in chlorosis and pernicious anæmia, the nutrition and function of all organs suffer, and the spinal cord shares the general state. The readiness with which fatigue of the legs is induced in such conditions may be due to the spinal cord as well as to the muscles. The legs often ache, and are sometimes the seat of various disordered sensations. It is probable that these are the result of the impaired nutrition of the nerve-elements, in consequence of which their functions are readily deranged. In some patients graver symptoms occur,—weakness of the legs, sometimes of the arms also, which may increase slowly to complete paralysis. Sensation and the sphincters are usually unaffected. The nature of these cases is uncertain. It is probable that many are cases of what is called hysterical paraplegia. Others may be due to organic changes induced by the anæmia. The well-established fact that optic neuritis may result from chlorosis is interesting as proof of the intensity of changes in nerve-structures which anæmia may excite. The cases of chlorotic paraplegia are very rare, and need further study.

In the anæmia that results from loss of blood, besides the symptoms just described, paraplegia sometimes comes on, under conditions which exclude the idea of hysteria. The source of the hæmorrhage has most frequently been the stomach, kidneys, and uterus. It is usually motor only, but Leyden has observed accompanying hyperæsthesia of the skin. It comes on sometimes soon after the loss of blood, sometimes only at the end of one or two weeks. Most cases recover. The pathological process which causes the paralysis is not known. The loss of power is comparable to the loss of sight which occurs from the same cause, and under similar conditions, and in which there may be no visible morbid change, or inflammation may be found, sometimes succeeding the loss of sight, and therefore to be regarded as a result of

the derangement of the nerve-elements, or of the influence to which this is due, rather than the cause of the amaurosis.

The treatment of the symptoms due to general anæmia is of course essentially the improvement of the blood-state. That of other symptoms will be considered in the account of functional disturbance of the spinal cord.

HYPERÆMIA OF THE SPINAL CORD.

The conditions of the return of venous blood from the cord probably shield it effectually from the mechanical congestion from which almost every other organ of the body suffers when the movement of blood through the thorax is hindered. The only mechanical congestion to which the cord is liable is that which results from the influence of gravitation, and occurs when a person is lying on the back. The distension of the veins outside the dura mater, and also of those of the pia mater, which occurs when the heart has ceased to act, and the blood is free to obey the only mechanical force which then acts upon it, sufficiently proves the power of gravitation to congest the cord. But it is not probable that gravitation has anything like the same influence during life, counteracted, as it then is, by many other forces. Some persons with weak cords (easily fatigued, liable to tingling and pains in the legs) suffer from aching in the legs or spine when they lie on the back. Since the discomfort ceases when the posture is changed, the pain is probably due to the mechanical congestion of the cord or membranes. It is doubtful whether any other symptoms can be, with confidence, assigned to mechanical congestion. Those other nocturnal disturbances in the limbs, that might conceivably be due to mechanical congestion, occur irrespective of posture.

Of active congestion our definite knowledge may be summed up in the statement that it occurs in the early stage of inflammation.* We *know* nothing of it as an independent condition. Nevertheless volumes might be filled by the collected descriptions of the varieties and symptoms of congestion of the spinal cord,—descriptions in which the unrestricted play of “scientific” fancy has elaborated a symptomatology for the separate congestion of every part of the spinal cord. Probably the only case in which we are warranted in suspecting a primary active congestion of the cord or membranes is that in which symptoms identical with those of commencing myelitis, and following a cause of myelitis, pass away in the course of a few days. These cases will be mentioned in the account of inflammation of the cord.

An active congestion, which may be called secondary, seems to result from prolonged and violent action of the nerve-elements of

* It may be remarked that the term “congestion” has come to be a popular medical euphemism for a slight degree of inflammation, employed because the public have become acquainted with the word as signifying a less grave lesion than inflammation, and hence the word excites less alarm. Such a use of the word, within limits, is less objectionable than that of many analogous euphemisms, but it is often grossly abused.

the spinal cord. Thus local dilatation and distension of vessels, migration of leucocytes into the sheath and into the adjacent tissue, and even small extravasations, are found in some cases of hydrophobia, of tetanus, and of strychnine poisoning, and it is probable that a similar congestion attends all violent physiological activity, violent and continued muscular exertion, coitus, &c. Coitus several times repeated has been known to cause hæmorrhage within the cord. Vascular dilatation, with an increased blood-supply, is the physiological attendant of functional activity in all organs, and doubtless also in the cord, but it is morbid only when excessive. It is doubtful whether it causes symptoms unless it goes on, as it probably may, to actual inflammation.

INFLAMMATION OF THE SPINAL CORD; MYELITIS.

Inflammation of the spinal cord is a common lesion. It usually causes a considerable diminution of consistence of the cord. Hence the term "softening of the spinal cord" has been used as a synonym for myelitis, but inaccurately, because inflammation does not always cause softening, and a diminution of consistence may occur without inflammation. The spinal cord differs from many other tissues of the body in that its power of recovering functional integrity, after damage by inflammation, is much less than that of many other organs. The damage produced, and the symptoms which result from that damage, persist for a long time, and are not unfrequently permanent. Hence the distinction between acute and chronic inflammation is to be drawn, not from the whole course of the disease, but from the mode of onset. It is this which indicates the character of the morbid process. *Acute myelitis* is that form in which the symptoms indicating inflammation of the cord come on rapidly and reach a considerable height in a short time, in less than two weeks. When the onset occupies from two to six weeks the inflammation is regarded as *subacute*. When a still longer time elapses before the symptoms reach a considerable degree of intensity the myelitis is termed *chronic*. There is, of course, no sharp distinction between these classes. The subacute and acute forms may be conveniently described together. Other varieties are based on the distribution of the disease. The grey matter may be affected only or chiefly, *polio-myelitis* (Kussmaul, from *πολιος* grey), a form that has distinctive characters which make its separate description desirable. When the whole thickness of the cord is affected in a small vertical extent the myelitis is said to be *transverse*. This is one of the most common forms. When an extensive area of the cord is inflamed the myelitis is said to be *diffuse*; when one small area is affected the inflammation is called *focal*; when there are many foci in various parts of the cord the

myelitis is said to be *disseminated*. Inflammation of the grey matter, around the central canal, extending into the intermediate grey substance and to a less extent into the cornua, has been termed *central myelitis*. It is said to be sometimes so intense that the grey substance flows out on section, leaving the white substance intact. From this the inflammation of the cornua (commonly limited to the anterior) may conveniently be distinguished as *cornual myelitis*. Both are forms of polio-myelitis, but the latter term has been somewhat anomalously restricted to the affection of the anterior cornu. To this it was originally applied, with the qualification "anterior," but the adjective is now frequently omitted.

ACUTE MYELITIS.

ETIOLOGY.—Little is known of the influence of hereditary predisposition, but myelitis is sometimes met with in members of families in which other evidence of a neuropathic tendency can be traced. Males suffer more frequently than females. Acute myelitis may occur at all ages, but the form which occurs in young children is usually confined to the anterior cornua; other forms of myelitis are most common between ten and forty years. Of exciting causes the most frequent is exposure to cold, especially to wet cold,—exposure to cold winds when the body is heated, lying on damp grass or on snow, bathing in cold water. This cause has been supposed to act by suppression of the functions of the skin, since myelitis has been produced by varnishing the skin of animals,* but it is unlikely that this is the sole mechanism. Exposure of the back to cold sometimes seems especially effective. Myelitis has been produced experimentally in rabbits by freezing the skin of the back with ether spray (Feinberg). Over-exertion is another powerful cause, and is especially effective when combined with insufficient food, and exposure to cold. Hence soldiers on the march are often attacked. The disease has been known to follow a single violent contraction of the dorsal muscles. It followed, in one recorded case, a violent effort to prevent a fall on the ice. It is possible that there is an actual injury to the cord in these cases, or inflammation may be set up in the vertebral column and may spread to the cord. In several cases it has resulted from attempts to lift heavy weights. Thus a man, two days after such an attempt, became rapidly paraplegic and ultimately died, and softening of the cord was found at the level of the fifth and sixth cervical vertebræ. Sexual excesses, and the sudden suppression of the menses and of other habitual discharges, have been supposed to cause the disease.

Myelitis, acute and subacute, often results from injuries to the cord of all kinds, lacerations, bruises, and punctured wounds. The readiness with which traumatic myelitis may be produced, has been repeatedly demonstrated experimentally. Simple concussion of the cord may set

* Feinberg, 'Virchow's Archiv,' Bd. 59. In Vulpian's laboratory similar experiments failed.

up inflammation, but the subacute form most frequently results from this cause, and often follows railway accidents. Slow compression is constantly attended by inflammation in the compressed part, as stated in the account of compression; the myelitis thus set up may have an independent course, acute or subacute, and may not be proportioned to the pressure. In cancer of the vertebral bones, for instance, very slight pressure may cause an acute inflammation; there is probably sometimes an extension of inflammation to the cord from the external disease, but such extension is not necessary for the occurrence of the myelitis; the inner surface of the dura mater is often normal. Acute myelitis also results from internal meningitis, in which some degree of invasion of the cord is almost invariable.

Inflammation of the cord occasionally comes on in the course of, or after, some acute diseases, typhus, typhoid, and especially variola, acute rheumatism, and severe puerperal diseases. Whether syphilis is a cause of acute myelitis is a question on which opinions differ. It certainly may cause a subacute form. Attacks of acute myelitis are not uncommon in syphilitic subjects. It seems on the whole probable that there is a causal relation between the two, although the nature of that relation, whether, for instance, it is by disease of the vessels, is not known. Subacute myelitis may result from chronic alcoholism, but it is doubtful whether acute myelitis is produced by this cause.

Irritation and inflammation of peripheral organs, as the uterus, bladder, kidneys, intestine, &c., has been supposed to be an occasional cause, but this influence is exceedingly doubtful except in the rare cases in which inflammation of a nerve ascends to the cord. In a considerable number of cases we are able to discover no cause to which myelitis can be ascribed.

PATHOLOGICAL ANATOMY.—In recent, acute inflammation of the cord, the pia mater, at the affected part, is usually red and vascular, and the cord itself may be slightly swollen. Its tissue is reddened and often marbled from distended vessels, and the distinction between white and grey matter is indistinct and lost.

The diminution of consistence which occurs in acute inflammation may be so great that the affected part is diffuent, like cream, and flows out when the pia mater is divided. It does not follow that the consistence is lessened to this degree during life; post-mortem softening occurs more readily at an inflamed part. When the vascular disturbance and extravasation are considerable, the condition is termed "red softening:" so much blood may be effused into the tissue that the part looks as if there had been a primary hæmorrhage. This form is termed "hæmorrhagic myelitis." If the inflammation has existed for some time, the effused blood is altered in tint and the colour of the softened part may be rather chocolate than red. After a longer time the changes in the blood-pigment cause the colour to be lighter, "yellow softening." Ultimately the blood-pigment is removed and the

softening becomes "white." The period at which these changes of colour are found varies according to the amount of extravasation at the onset. This is sometimes very slight, so that the softened part may be white from the first. At a later period the white, opaque aspect is changed to a grey translucent appearance, from the absorption of the fatty products of degeneration, and an increase in the connective tissue. If the myelitis is very limited in extent, cavities may be formed within the cord. When the destruction of tissue is trifling, the diminution in consistence may be very slight.

The microscopical appearances in myelitis vary much according to the duration and form of the inflammation, and also according to the method of preparation adopted. Observations during the early stage are scanty, since few patients die during the first fortnight. Moreover, the softened tissue does not harden, so that the most diseased part cannot be examined by the most instructive method. In the fresh state the most conspicuous objects are the products of degeneration of the fibres, granules and masses of myelin, and granule corpuscles, with, in older cases, the peculiar bodies termed "corpora amylacea" (Fig. 84, *d*). With these are numerous red blood-discs, leucocyte-like corpuscles, and, after the first week, distinct cells, round, oval, spindle-shaped, or angular, with nuclei which resemble leucocytes in size, and are readily mistaken for them. Fragments of axis-cylinders may also be seen, granular in aspect, and with irregular swellings (Fig. 84, *a*). After hardening, the appearances vary according to the intensity of the inflammation. The changes in the acute and sub-acute forms are very similar, probably because, in the parts that can be thus examined, the acute inflammation is less intense than in the softened parts, and because acute cases usually come under pathological observation when the acute stage is over. The products of degeneration are best seen in the fresh state or in sections mounted in glycerine (Figs. 84 and 99). The fragments and globules of myelin in part occupy the position of the nerve-fibres from which they have come,



FIG. 84.—Myelitis. *a*, An irregularly swollen axis-cylinder. *b*, Section of nerve-fibre with swollen axis-cylinder. *c*, Section of white substance, with granule-masses and atrophied nerve-fibres. *d*, Corpora amylacea.

in part are aggregated into masses. In sections rendered transparent in the ordinary way, the most conspicuous alteration is the dilatation of vessels, which is often very great. In the white substance it is chiefly the larger vessels that are enlarged, but the grey substance may be crammed with dilated vessels (Figs. 85, 86, A). The nuclei of the capillaries are enlarged. The walls of the smaller arteries are encrusted with leucocytes in the early stage, and, later on, in both white and grey substance, are much thickened by cells, the nuclei of which are often elongated and more or less concen-

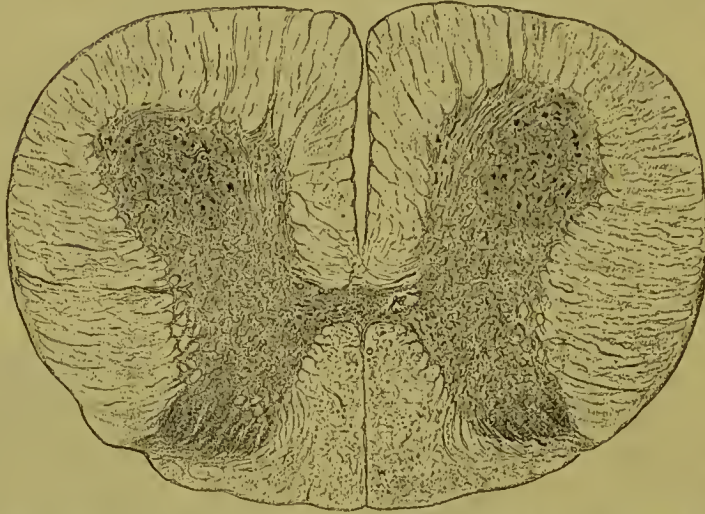


FIG. 85.—Subacute myelitis, lumbar region; both white and grey substance uniformly affected. In the latter the numerous distended vessels appear as lines; here and there a larger one is visible.

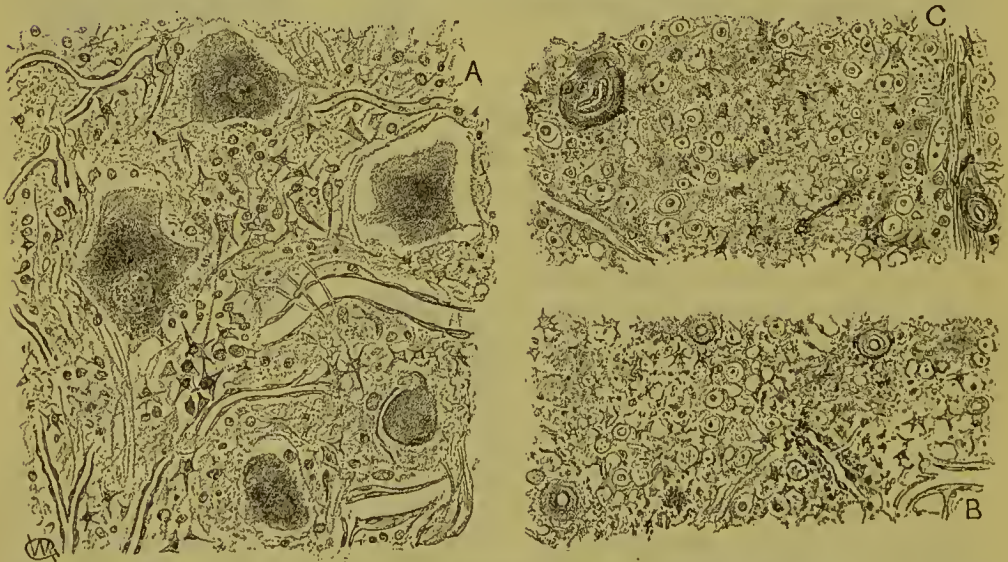


FIG. 86.—Portions of the section shown in the last figure, more highly magnified. A, from the left ant. cornu; distended capillaries with numerous nuclei; larger vessels with slight distension of perivascular sheaths; numerous round and stellate cells; ganglion cells, swollen, with shrivelled processes. C, from front of post. column; scattered nerve-fibres separated by amorphous material in which small cells can be seen, round and irregular, some distinct, others indistinct, vessels with thickened walls. B, from near the posterior surface, shows similar changes but with more open spaces from which nerve-fibres have perished. (From sections prepared by Dr. Money.)

tried to the lumen of their vessel (Fig. 89). Outside this thickened wall the perivascular sheath is enormously distended, at first by leucocytes, among which, afterwards, other cells are mingled, round, fusiform, angular, with leucocyte-like nuclei (Fig. 89). In transverse section the distended sheath may look, at first sight, like an enormously thickened wall. Red blood-corpuscles distend the vessels and are seen also in the adjacent tissue, sometimes uniformly scattered through it (Fig. 89), sometimes aggregated in small extravasations. The grey substance is densely set with round corpuscles, staining deeply, many of which are the nuclei of small, fusiform, or angular cells (Figs. 86, A ; 88, B), while the intervening substance is much more granular than in health. The nerve-cells are much swollen and granular, often contain distinct globules, strongly refracting, and probably fatty. The cell-processes appear shrivelled or lost (Fig. 86, A). In slight cases the margins of the cells may be less sharply defined than in health, and the bodies then may contain large vacuoles, sometimes wholly within the cells, sometimes partly within and partly outside them in an adjacent albuminous-looking substance.*

In the white substance cleared sections show a great increase and alteration in the interstitial tissue. The change may be uniformly distributed, or greatest in the neighbourhood of the vessels (Fig. 89, c). The increase is due in part to an amorphous material, containing nuclei here and there, some of which belong to cells such as have been described in the grey substance; and these may constitute extensive tracts between the spaces where nerve-fibres once existed (Fig. 89). In older cases the tissue may have a fibrous aspect in places. The large cells, with many processes, called "spider cells" or "cells of Deiters," are often conspicuous objects (Fig. 87). The processes extend between the nerve-fibres. We have seen (p. 107) that their existence in the normal cord has been doubted, but in the inflamed cord, in which they are enlarged, their presence is unmistakable. The changes in the nerve-fibres vary much. They always suffer destructive changes, but the products of their degeneration, above described, are scarcely to be seen in cleared speci-

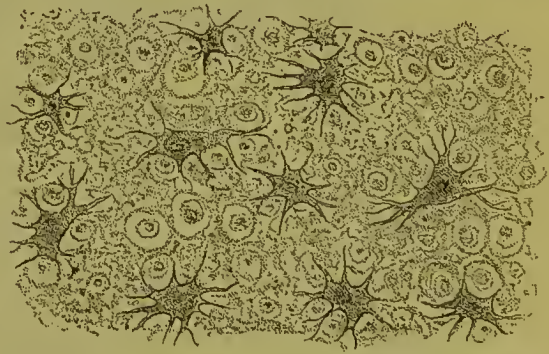


FIG. 87.—Subacute disseminated myelitis; part of the posterior column. Nerve-fibres separated by granular material and products of degeneration, and by numerous large spider cells. The myelitis in this case was attended by optic neuritis. (From a section lent by Dr. Dreschfeld.)

* The significance of this vacuolation has been much discussed. It is probable that the vacuoles form after death, perhaps during the process of hardening, but that they do not form unless the cells have been altered by disease. They have thus some pathological significance.

mens. The slightest change in the myelin appears to consist in an alteration in its chemical nature, in consequence of which it stains more readily than in health. Fibres are thus changed adjacent to spots of increase in the interstitial tissue, and they contrast with the unstained

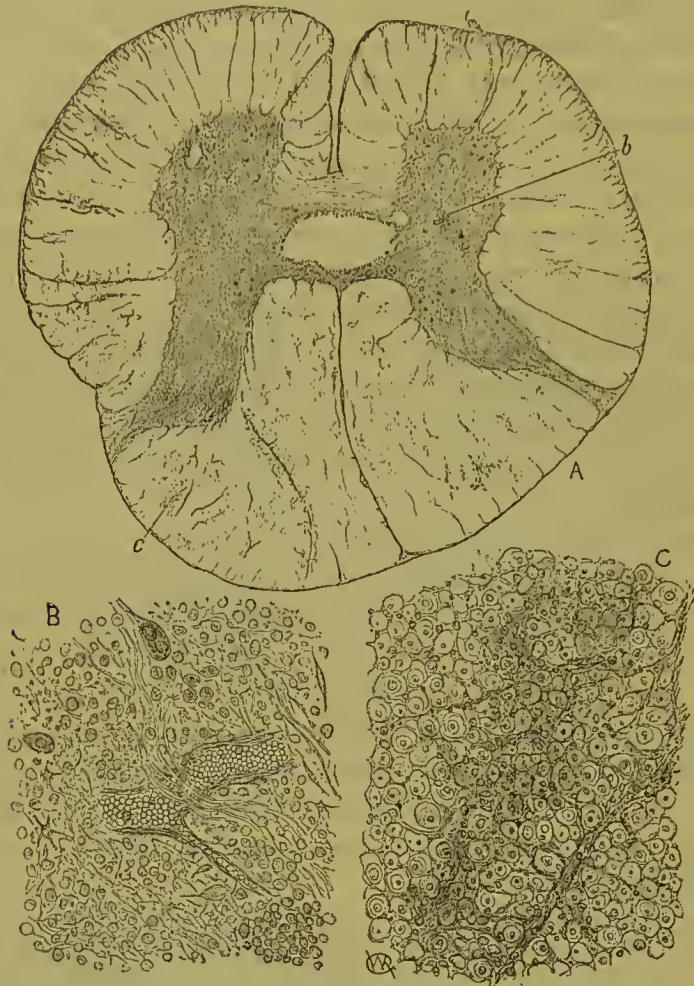


FIG. 88.—Acute transverse myelitis, fatal in three weeks. Carmine preparations. A, Section through most diseased part, at first lumbar segment. Foci of inflammation scattered through the whole area of this cord, most abundant in the posterior columns (the apparent enlargement of which is probably due to the direction of the section). Dilatation of the central canal. B, From the anterior cornu at A, *b*, a distended vessel; the tissue crammed with lymphoid and other cells, among which run normal nerve-fibres. C, From the posterior column, A, *c*. The areas staining deeply are seen to consist of thickened trabeculae, lymphoid cells, amorphous tissue, and also of nerve-fibres, the white substance of which stains with carmine while that of the neighbouring healthy fibres does not.*

healthy fibres in the vicinity (Fig. 88, *c*). With or without this change in the myelin there is an irregular swelling of the axis-cylinders, such as is seen on examination in the fresh state. If the fibres are divided

* I am indebted to Dr. Dreschfeld for the section from which these drawings are made. The patient was a girl aged twenty-three; paralysis, motor and sensory, was complete at the end of two days; the application by her mother of hot bottles to the feet caused such extensive sloughing ulcers that amputation of one leg was deemed necessary three weeks after the onset, and the patient died the next day.

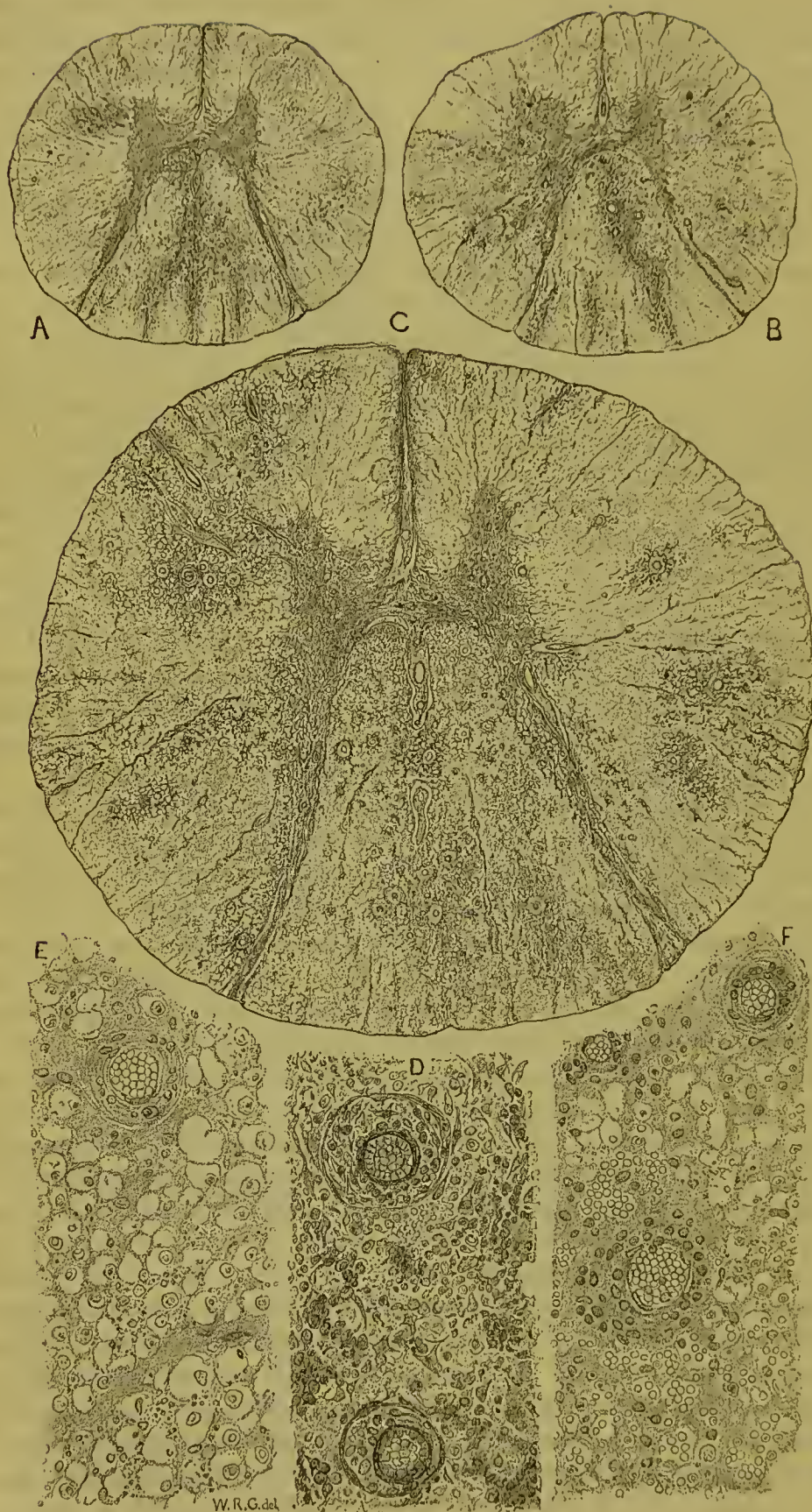
transversely at the places where the axis-cylinder is swollen this appears much larger than normal (Fig. 84, *b*). Examples of this will be found in most of the figures. These intense degenerative changes cause the breaking up of the myelin and may involve the destruction of the fibre, the space occupied by it being left empty. In other cases, however, the axis-cylinder persists and remains surrounded by a narrow zone of myelin. Often a large number of such shrunken fibres may be found in a part which at first sight appeared devoid of fibres. This shrinking of the fibres occurs not only as a result of pressure on them by the connective-tissue elements, but also where there is little interstitial change and no compression. The relative amount of change in the nerve-fibres and interstitial tissue respectively varies very much. In some cases the interstitial increase of tissue seems the primary change and the fibres are altered secondarily, changing, shrinking, and vanishing as they are surrounded and enclosed by the new tissue.

In other cases, or even other parts of the same section, there is but little increase in the neuroglia although the fibres have suffered extensively; empty spaces are limited by septa but little thicker than normal, although with more nuclei and often with numerous granules, the nature of which is uncertain. Occasionally there is a more diffuse change in the white substance; the neuroglial tissue is increased by small cells and amorphous material in such a manner that all appearance of septa and spaces is lost; a few nerve-fibres are included in it, and the spider cells are very conspicuous objects, their processes alone suggesting the septa that exist in the normal condition (Fig. 87).

The distribution of these changes is very variable. The whole thickness of the cord is uniformly affected in pressure-myelitis (see "Compression of the Spinal Cord"). In transverse myelitis the alteration is rarely uniformly distributed through a single section, and each part may present variations in the damage in a series of sections taken near together (see Fig. 89). In meningo-myelitis the periphery of the cord is always most affected, and in old cases the thickened pia mater may send tracts of dense tissue into the interior of the cord, from which branching processes of tissue may pass on all sides. An illustration of this is given in the section on chronic myelitis. A similar extension is often conspicuous in cases in which there is no special inflammation of the membranes, and the interstitial changes often extend from the septa that normally pass into the cord and from the walls of the vessels, the course of which may thus be marked out when the vessels cannot be seen. Hence the white columns may be broken up by lines of tissue passing from the surface of the cord towards the grey substance. In disseminated myelitis foci of inflammation may be scattered through a wide extent of the spinal cord and may appear on the surface as reddish-grey areas, closely resembling those of disseminated sclerosis, but less sharply limited. This form is usually subacute and is that which is often due to syphilis.

The nerve-roots coming from a much inflamed part are usually

FIG. 89.



damaged, and may present histological changes similar to those within the cord, distension of vessels, increase of nuclei and of connective tissue about the vessels, breaking up of the myelin, swelling of the axis-cylinders. Degenerative and neuritic changes may descend the motor nerves, although rarely to the same degree as in polio-myelitis.

From the foci of inflammation, ascending and descending degenerations pass along the tracts of long fibres that have been already described, downwards in the pyramidal tracts, upwards in the posterior median columns, the direct cerebellar tracts, and the anterolateral ascending tract. In cases of transverse myelitis inflammation sometimes ascends the cord for a short distance chiefly in certain tracts. It may, for instance, pass up the pyramidal tracts, so that, an inch or two above the upper limit of the general inflammatory changes, an ascending inflammation of the pyramidal tracts may be combined with an ascending degeneration of the tracts that always suffer secondary ascending degeneration.

If the patient survives, the new interstitial tissue slowly undergoes fibroid changes and contracts, although for a long time cell-forms may be distinguished in it. We know very little of the nature of the process that occurs in the cases which slowly recover. The fact that a long period of total palsy may be succeeded by the slow return of considerable power shows that greatly damaged fibres may regain functional capacity. It is easy to conceive that the fibres which are only so far changed, that the axis-cylinders are swollen and the myelin stains readily, may speedily recover. The fibres that are shrunk, and in which a very narrow layer of myelin surrounds an axis-cylinder, may also regain the power of conducting. But we must presume that in these fibres there is no interruption of the axis-cylinders. An absolute motor palsy of twelve months' duration must, it would seem by all analogy, involve an absolute interruption of the fibres. But in such a case some return of power may occur. Is this effected by a growth of new fibres such as occurs in nerves? In the lower animals such a growth of fibres has been proved to occur; in man it has not yet been demonstrated. But it is difficult to conceive any other explanation of the clinical facts. The very interesting appear-

FIG. 89.—Acute transverse myelitis; from a case fatal in a month. A, B, C, Neighbouring sections from the inflamed part, in the mid-dorsal region, to show the varying distribution of the inflammation. (Stained with aniline blue black.) Distended vessels are all surrounded by thick zones of clear tissue, in places divided obliquely, while outside this, ramifying tracts of darker tissue extend into the white substance. In D, from the front of the posterior column, the changes are shown, more highly magnified. The zone around the vessels is seen to consist of the perivascular sheath distended by cellular elements, round, oval, fusiform, angular. The white substance is crammed with similar cells and hardly any trace of nerve-fibres or of normal structure can be perceived. In E, from a less affected part of the lateral column, stained with carmine, the alveoli remain, although the tissue between them is thickened, and in many places studded with refracting granules and larger nuclei. In many spaces nerve-fibres are seen much smaller than normal in consequence of the wasting of the white substance. F represents a similar condition, with fewer nerve-fibres, and extensive infiltration of the alveoli with blood-corpuscles. (From sections prepared by Dr. Moncy. For the specimen I am indebted to Dr. Hadden.)

ances presented by the section of a cord shown in Figs. 90 B and 91 suggests strongly a process of regeneration. The cord was crushed in the mid-dorsal region by a fracture of the spine, and motor and sen-

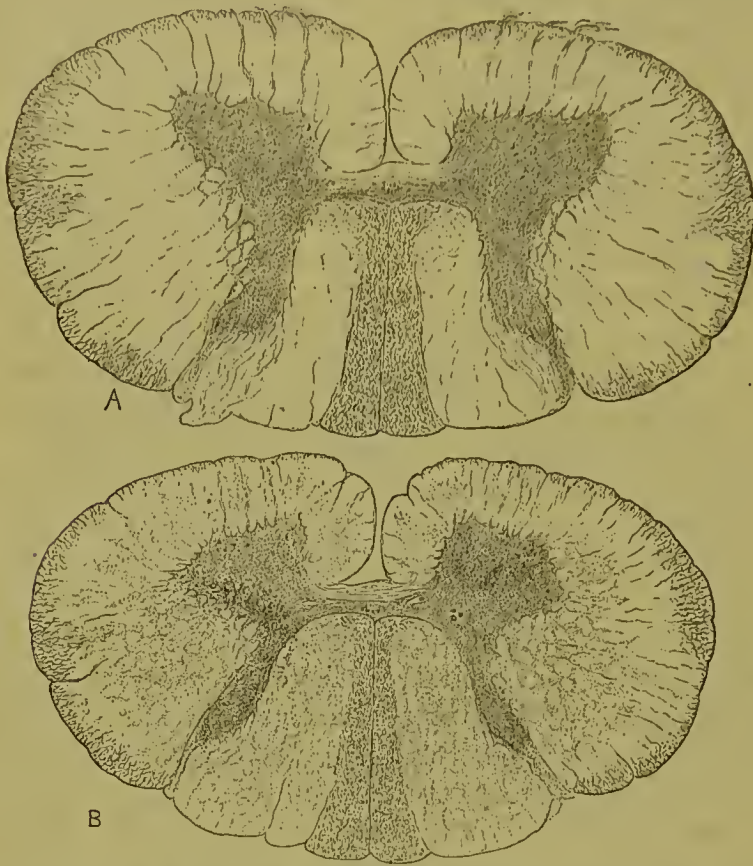


FIG. 90.—Ascending myelitis from fracture of spine completely destroying the cord in the mid-dorsal region. A, from the seventh cervical, presents only the ascending degenerations in post. med. col., direct cerebel. tract, and ant.-lat. ascending tract. B, from the eighth cervical, presents also numerous tracts of finely granular aspect under a low magnifying power, the structure of which is shown in Fig. 91.



FIG. 91.—From the outer part of the front of the lateral column in B, Fig. 90. *a* is the antero-lateral ascending tract, the fibres completely degenerated, except a few, probably of other nature and belonging to *b*, a narrow zone, nearly healthy, of the antero-lateral ground fibres. *c* represents the outer part of one of the finely granular tracts. Under the higher magnifying power these tracts are seen to consist of very minute nerve-fibres with a granular material between them in which few cell-forms can be distinguished. Adjacent to these tracts there has apparently been extensive destruction of the fibres, large spaces being occupied by products of degeneration and appearing empty in the cleared section.

sory paralysis remained absolute, up to the level of the lesion, till the patient's death, six months later. At the seventh cervical the cord presents only the usual ascending degenerations, but at the eighth cervical, opaque tracts extend through the grey matter. Adjacent to these there is everywhere an extensive destruction of nerve-fibres. The tracts contain a large number of very minute nerve-fibres, each consisting of a fine axis-cylinder surrounded by myelin. The fibres are smaller than any met with in the normal cord. The appearance is as if there had been an ascending myelitis, chiefly parenchymatous, which had extended up the cord as high as between the seventh and eighth cervical segments, and from the lower extremity of the normal fibres there had occurred a growth of new fibres such as effects the regeneration of nerves.*

SYMPTOMS.—The symptoms of myelitis vary much according to its degree and form. The most common variety, which may be regarded as the typical form, is acute transverse myelitis, and to this especially the following description applies. The conspicuous symptoms are those which depend on the interference with the functions of the cord, and these, in most cases, are also the first indications of the disease. They are sometimes preceded, and more often accompanied, by general symptoms, such as attend inflammation of other internal organs,—malaise, shivering, headache, depression, loss of appetite, pyrexia,—symptoms which may attend any form of acute myelitis, or may be altogether absent. Their occasional absence is probably explained by the fact that very severe nervous symptoms may result from an inflammation which involves a very trifling extent of tissue.

The spinal symptoms, if not prominent at first, quickly become so. The most obtrusive is the motor weakness, but this may be preceded, for a few hours or days, by sensory symptoms in the limbs—"numbness," tingling, formication, or burning. Occasionally there are rheumatoid pains at the onset, especially in myelitis of the anterior cornua. Pain may be felt in the back, as well as in the limbs, but the vertebral pain is not increased by movement, and soon ceases. Occasionally spasmodic twitchings in the limbs or painful cramps attend the onset. In rare cases, there is a general convulsion. This is most frequent in children, and then may be sometimes the expression of the general disturbance of the system. But convulsion occasionally attends the onset of acute myelitis in adults, even when there is no cerebral complication. Three instances of this have come under my own observation.

The motor paralysis usually comes on rapidly, and reaches a considerable degree in the course of a few hours. A patient after such sensations as have been described, or without any warning, finds that his legs feel heavy; after walking for a few hours he is obliged to sit down

* For these sections I am indebted to Dr. F. G. Penrose. Unfortunately I have been unable to obtain any other sections of this part of the cord.

and rest. When he tries to walk again his legs feel "as if made of lead." He lies down for an hour, and then finds that he cannot stand, and in a few hours more is unable to raise his legs in bed, although perhaps he can still move the toes; next day, even this power is lost. Occasionally the mode of onset is still more rapid, and occupies only a few minutes. The legs are found suddenly to be heavy and tingling, the sufferer sits down on a chair for a quarter of an hour, and then finds that he cannot stand. Such rapid onset resembles that of spinal hæmorrhage, and it is probable that in most of these cases there is hæmorrhage in addition to inflammation, "hæmorrhagic myelitis." Such cases are, however, sufficiently rare not to interfere with the diagnostic rule that a sudden onset means a vascular lesion, and that the characteristic onset of myelitis is rapid but not sudden. Now and then the onset is in the night, during sleep; a patient goes to bed well, and wakes up in the morning with complete paraplegia. On the other hand the onset of the paralysis may occupy several days, or even a week. When longer than this, from ten to forty days, the myelitis is to be regarded as subacute. Occasionally the onset is by a series of sudden attacks of weakness at intervals of a few hours or days, or even weeks. In the latter case there may be some recovery from one attack before the next comes on. In other instances, there is gradual partial loss of power for a few days, and then complete paralysis comes on rapidly.

When the paralysis has reached its height, and has ceased to increase, it is usually complete; sometimes it is incomplete, the limbs can be moved, but with little power, or the patient may be able to contract certain muscles but not to move the parts to which they are attached, perhaps only to move the toes. The distribution of the paralysis depends on the position and extent of the disease. In the majority of cases the legs and lower part of the trunk only are affected, because the dorsal region of the cord is the most frequent seat of myelitis. If it is incomplete in the legs, the flexor muscles usually suffer more than the extensors. When the disease is in the cervical region the arms as well as the legs are involved, and the arms are sometimes paralysed before the legs, probably because the disease, in these cases, begins in the grey matter, and affects the pyramidal tracts secondarily (Hallepeau). The range of motor symptoms, when the disease is at different heights, are indicated in the table of functions given at p. 142.

Sensation is frequently impaired as well as motion. In all cases, except those of trifling degree, it is affected at the onset. It may be absolutely lost up to the level of the lesion. In slighter cases the loss may be partial, and only certain forms of sensation may be impaired. Occasionally there is hyperæsthesia in the limbs at the onset. At the level of the lesion, there is usually a zone of hyperæsthesia, corresponding to the distribution of the nerves that pass through the upper part of the affected region, *i. e.* through the lowest, merely irritated, portion of the cord above the disease. The hyperæsthesia may readily

be detected by passing a hot sponge down the spine; the sense of warmth changes, at the hyperæsthetic zone, into one of pain. Corresponding to this zone of hyperæsthesia there is often a sense of painful constriction, a "girdle pain," which may persist for a long time, even after the loss of sensibility below the lesion has passed away. Its position varies with the position of the disease. It is most commonly felt between the umbilicus and ensiform cartilage, sometimes around the lower part of the abdomen, about the anus, or in the legs. It is of much practical importance because it is evidence of the existence of organic disease and also of the upper limit of the lesion. It is sometimes a very early symptom. Any initial pain in the limbs usually ceases when sensation becomes abolished.

The state of reflex action varies, and depends on the position of the disease, in accordance with the laws already stated. An acute lesion in any part of the cord may cause an initial, inhibitory, loss of reflex action in the parts below, but if the lesion is above the lumbar enlargement, reflex action returns in the course of a few hours. Frequently there is no initial depression. Subsequently the reflex action becomes excessive, that from the skin rapidly, that from the muscles more slowly. Ultimately each attains a high degree of exaltation. If the disease involves the lumbar enlargement all forms of reflex action are lost. When in the cervical region, it may be lost in the arms, excessive in the legs. In dorsal myelitis the trunk-reflexes are often impaired, and may give important information as to the seat of the disease.

The muscles of the legs are sometimes at first flabby and toneless during the stage of initial depression of reflex action, doubtless from the same influence. This condition soon passes off, if the lesion is above the lumbar enlargement, and as reflex action becomes active, the muscles regain their tone. If, however, the myelitis involves the lumbar enlargement in considerable degree, the muscles of the legs

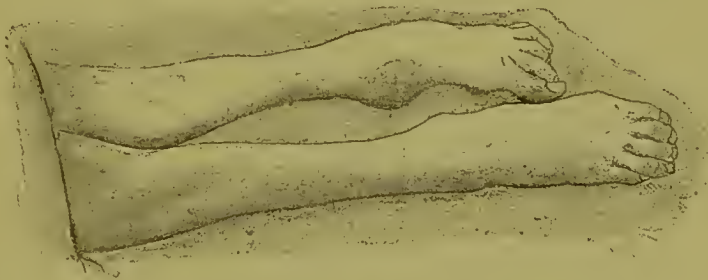


FIG. 92.—Posture of feet in myelitis of lumbar enlargement with rapid wasting of muscles.

remain flaccid, and lose all influence on the posture of the limbs. The feet fall into an extended position, so that the instep is in a line with the tibia (Fig. 92). The muscles also waste rapidly, and present the reaction of degeneration. The wasting in these cases is often extreme.

In many cases of myelitis above the lumbar enlargement there is a slighter and slower wasting of the muscles, without any loss of reflex action. The ultimate amount of wasting may be considerable, but it is never so great as when the lumbar grey matter is diseased. There is never the reaction of degeneration, but only a slight change in irritability, similar to each current, at first an increase which, after some weeks or months, gives place to a slight diminution.

The sphincters are usually affected from the first, except in very slight cases, and they sometimes afford the earliest indication of commencing lesion. There is usually first retention of urine, and afterwards incontinence, overflow or reflex (see p. 153), when the disease is above the lumbar region; if the lumbar centre is diseased there is persistent incontinence from the first. In the former case there is involuntary action, in the latter inaction, of the sphincter ani, and in both cases incontinence of fæces. This may be inconspicuous in the early period, on account of constipation, due to intestinal paralysis. In cases of partial myelitis, as of the anterior cornua, the sphincters are often unaffected. The urine is frequently alkaline in reaction, and the change sometimes comes on so rapidly that it is evidently the result of an alteration in the secretion. In all cases of retention, decomposition in the bladder increases the alkalinity. Cystitis often follows.

The temperature of the paralysed limbs is usually at first raised one or two degrees above that of the mouth, but it afterwards falls, and remains a degree or so lower than that elsewhere. The skin is often dry, sometimes covered with sweat. Its nutrition often suffers and bedsores result. In some cases they are due to long-continued pressure and neglect, but they may occur early and intensely when the disease affects the lumbar enlargement or irritation extends down to it from above. The least pressure on the skin may then cause a bulla containing sanious liquid, and sloughing occurs with readiness, especially over the sacrum. Now and then, there is effusion into the knee-joints. Occasionally the tendency to sloughing of the skin is so intense that the lesions appear to be spontaneous.

Such trophic disturbances sometimes occur in the cellular tissue of the lower part of the body, especially in the neighbourhood of the bladder and rectum. A remarkable example of such disturbance was presented by the case from which Figs. 85 and 86 are taken. Slight symptoms of subacute myelitis were followed by those of cellulitis in the lower part of the abdomen, on account of which the patient was admitted into University College Hospital under the care of my colleague Mr. Marcus Beck. Subcutaneous emphysema developed about the groins, and suppuration occurred near the rectum. In consequence of this inflammation the patient died, the cord symptoms having slowly increased. After death no lesion of the intestine or local cause of the cellulitis could be discovered. It is probable that the derangement of the trophic influence of the cord facilitates the occurrence of cystitis

from retention of urine, and intensifies its effect on the kidneys. Ulceration of the bladder, which sometimes occurs with remarkable rapidity, may be thus produced. A vesico-vaginal fistula may form. Ulceration of the urethra, and consequent extravasation of urine have been observed in a similar case.* In another case the inflammation of the bladder was intense, and suppurative cellulitis occurred outside the organ and set up a fatal peritonitis. The kidneys were the seat of very acute suppurative inflammation†

When the disease is in the cervical region the pupil may be affected. In rare cases of myelitis, optic neuritis has been observed, without any intracranial complication to cause it.‡ It is probably not the result of the inflammation of the spinal cord, but is an associated and similar lesion, the result of the cause of the myelitis. It is noteworthy that most of the cases thus accompanied have been instances of disseminated myelitis, a form that suggests a cause acting widely on the nervous system. In the case of Sharkey and Lawford the optic neuritis reached its height some weeks before the occurrence of the first spinal symptoms, and in the cord were two separate and distant foci of inflammation.

In cases of rapid development the degree attained by the symptoms at their onset may not be exceeded. In some cases a slower increase or extension ensues in the course of a few days. The myelitis, beginning below, may ascend the cord, and the arms and respiratory muscles may gradually become involved. Or inflammation, beginning in the dorsal region, may slowly descend into the lumbar enlargement, probably extending along the grey matter. Reflex action in the early stages is normal or excessive, and afterwards becomes lost.

If the patient survives, the course of the symptoms varies according to the intensity of the lesion. The paralysis, motor and sensory, may remain complete. More frequently sensation is recovered after a few weeks or months, while motor power continues absent for a much longer period. The excessive reflex action may lead to the gradual development of spasm in the legs. This is especially related to the increase of the muscle-reflex action, which follows the secondary descending degeneration of the pyramidal fibres. Ultimately the condition is that termed "spastic paraplegia" and described in detail in another section. The onset of this spasm is not incompatible with the recovery of some voluntary power. The muscles in these cases are often well nourished and may even increase in size; the spasm constitutes a powerful stimulus to their growth. Spasm may however, coexist with the slow wasting described above, (p. 226), but is rarely intense in these cases. Sometimes, especially when the cuta-

* S. West, 'St. Bart. Hosp. Rep.,' vol. x.

† Sharkey and Lawford, 'Trans. Ophth. Soc.,' 1883.

‡ Optic neuritis was present in the case of disseminated myelitis under the care of Dr. Dreschfeld from which Fig. 87 is taken, and also in that of Sharkey and Lawford just mentioned. See 'Medical Ophthalmoscopy,' 2nd ed., p. 161.

neous reflexes are in excess, and there is little or no recovery of voluntary power, flexor spasm comes on at a later period, at first in paroxysms, alternating with the extensor spasm. Afterwards posture may lead to persistent contraction in the flexors of the knee and hip



FIG. 93.—Flexor contraction of legs in myelitis of the dorsal region.

(Fig. 93), which may become so great that the heels are in contact with the buttocks.

Death may occur from the extension upwards of the mischief, or by failure of respiratory power, often aided by accumulation of mucus in the bronchi, or it may result from the trophic consequences of the disease; bedsores may lead to pyæmia; cystitis and retention may set up suppurative pyelo-nephritis; or the patient's strength may be slowly worn out, and thus he may die after some weeks or months. When improvement takes place it is usually slow. In cases of moderate severity the recovery may ultimately be complete, but in severe cases it is often imperfect, and some weakness with spasm or wasting is permanent. Rarely there is no improvement. The disease passes into a chronic stage, and the paralysis may remain complete as long as the patient lives, often for years. In some cases, however, improvement occurs even after the paralysis has remained absolute for six or twelve months. In such cases the recovery is never complete, although it is sometimes considerable.

VARIETIES.—The different forms of myelitis, which may be distinguished on pathological grounds, are attended by some differences in the symptoms. The symptoms of *transverse myelitis* have been described as those typical and characteristic of the disease. Strictly speaking, the transverse form is a variety of *focal myelitis*, but the latter term is often applied to the variety in which there is a small area of inflammation not extending through the cord and sometimes limited to a single element. Many cases of anterior polio-myelitis are instances of this form, but these are excluded from present considera-

tion. With this exception, little is known of myelitis limited to a single small focus, because such cases are rarely fatal. Cases are occasionally met with in which symptoms of very limited range come on acutely, and may reasonably be ascribed to such a lesion. They may be one-sided, and motor or sensory, or, as I have more than once seen, there may be acute sensory disturbance with inco-ordination, limited to a single limb. A much more frequent focal form is *disseminated myelitis*, in which there are two or more foci of inflammation in the same or different parts of the cord. The onset of this form is often subacute, and constitutional symptoms are frequently absent. The most important special symptoms are those which indicate interference with the central functions of the cord in more than one locality. The combinations of symptoms are very variable. In many cases the several foci of inflammation develop successively, not simultaneously, and then we have a corresponding succession of symptoms. Thus, a myelitis in the cervical region causing atrophy of some groups of muscles in one arm and paralysis of the corresponding leg, may be followed by paralysis of the other leg with such loss of reflex action in it as shows a separate focus of inflammation in the lumbar enlargement, and this again by the development of a girdle pain corresponding to the middle of the dorsal region from a fresh area of inflammation in that situation. When numerous foci of myelitis occur in the dorsal region, as is not uncommon in syphilitic cases and after injury, the symptoms may closely resemble those of a transverse myelitis, because by one or another of these foci each of the elements of the cord is interrupted. The only distinction may be the extensive impairment of the trunk-reflexes, or the detection of extensive loss of irritability in the trunk-muscles.

In the rare form of *diffuse central myelitis* there is usually rapid loss of power, of sensation, and of reflex action, considerable elevation of temperature, speedy trophic disturbance, and often death at the end of two or three days. Rarely sensation and reflex action have been lowered and not lost. On the other hand, the loss of sensation is said to be sometimes absolute, when motor palsy is incomplete. The symptoms have begun in arms and legs simultaneously, or in either of these, and have accordingly spread upwards or downwards. Practically nothing is yet known of any slight non-fatal forms of this variety. *Hæmorrhagic myelitis* is scarcely a special form, since any acute inflammation of the cord may be attended by a sudden extravasation of blood. Its manifestation is the sudden onset of severe symptoms after slighter disturbance such as indicates a commencing myelitis. XX

PATHOLOGY.—Very little is known of the pathology of acute myelitis, of the relation of the morbid process to its causes. The spinal cord differs from most organs in that acute inflammation may be very limited in extent. In its great liability to primary inflammation, the cord presents a remarkable contrast to the brain. The

special liability of the dorsal region may be associated with the readiness with which this part undergoes post-mortem softening, but we are ignorant of the conditions by which this liability is determined. A question of considerable interest is whether acute inflammation of the cord always begins as such, or whether, in any case or form, it is set up by vascular obstruction, such as thrombosis in a minute vessel. It is conceivable that such an initial lesion may be ultimately lost in the spread of the inflammation it excites. The acute inflammations of the cord that occur in syphilitic subjects seem to have no specific histological characters.

DIAGNOSIS.—Myelitis is recognised by the rapid onset of symptoms indicating structural disease of the cord. The diagnosis may be aided by the presence of such general symptoms as attend the occurrence of inflammation in other organs, but the absence of these is of little negative significance. The position of the myelitis must be inferred from the considerations already described. Its upper limit is indicated by the upper limit of the paralysis; its lower limit by the impairment of the functions of the cord as a central organ (reflex action and muscular excitability) in the parts paralysed, while the degree to which the various structures of the cord are damaged must be inferred from the character and degree of the symptoms in the affected limbs.

Thus if called to a patient who has rapidly become paraplegic, the practitioner should first note the degree of motor and sensory paralysis of the legs (indicating whether the lesion is total or partial), and how high up the trunk the symptoms extend. He should then test the reflex action in the affected limbs and trunk, and ascertain the state of myotatic irritability in the limbs. These indicate the condition of the reflex arcs in the lower portion of the cord. Further information on this point is afforded by the state of muscular nutrition, and especially by the evidence of the state of nutrition of the nerve-fibres which is revealed by faradism. It is useless to apply this test until five or seven days after the onset, because four or five days, and often eight or ten, elapse before the degenerative changes occur in the nerve-fibres. The examination may then be made without any risk, provided a very gentle current is employed, just sufficient to cause a contraction in the corresponding muscles of a healthy limb, and the current need not be applied to each spot for more than a second. The isolated faradaic shock may be employed with advantage. If no change is found, the examination may be repeated at the end of ten days from the onset. If no muscles, at the end of that time, present any considerable diminution of contractility, it shows that no considerable nutritive change is taking place in the nerves, and that the grey matter from which the nerves proceed is not inflamed. On the other hand, if certain muscles present a diminution of irritability, others being normal, there is a focal lesion in the corresponding grey

matter. If all the muscles of both legs present such a failure there is an inflammation of the lumbar grey matter. So, in cases of cervical myelitis with paralysis of all four limbs, the condition of irritability of the arm muscles shows whether the disease involves, or is above, the grey matter in the lower half of the cervical enlargement. If there is impairment of irritability, its extent in the two arms will afford an indication as to whether the myelitis is total, or whether it affects chiefly certain spots in the grey matter (polio-myelitis). This distinction is aided in certain cases by the degree of paralysis of the legs. Cervical cornual myelitis may cause paralysis in the legs, because the inflammation, in slighter degree, involves also the lateral white columns, but the paralysis of the legs is usually incomplete, whereas it is complete if the myelitis is transverse, and sensation also may be lost. The distinction of the other varieties of myelitis depends on their special symptoms which have been just enumerated.

If transverse myelitis has been diagnosed, the question should always be asked, Is it primary, or secondary, to compression? We have seen that myelitis may result, even in acute form, from pressure on the cord. The answer must depend on the discovery of any cause of compression. The spinal column should in all cases be most carefully examined, not merely once, but again and again. When the myelitis is developed there may be no signs of bone disease, and yet these may appear in the course of a few weeks or months. Or the myelitis may be preceded by slight symptoms indicating irritation of the membranes. Myelitis in a cancerous patient should always suggest a secondary growth in the spine. A woman, shortly after the removal of a cancer from the breast, became paraplegic. No evidence of bone disease could at first be found, but in a few weeks the vertebral column became distinctly enlarged from secondary cancer. The diagnosis from spinal hæmorrhage is chiefly by the mode of onset. In hæmorrhage the symptoms develop in a few minutes, and it is only in the cases of hæmorrhagic myelitis that any diagnostic difficulty presents itself. The sudden paralysis is then commonly preceded by slight sensory symptoms, tingling, &c., and sometimes by fever. If these are absent in cases of actually sudden onset, we are not justified in diagnosing myelitis.

Cases in which acute myelitis ascends the cord, so that the legs, muscles of the trunk, and the arms are successively paralysed, bear considerable resemblance to the rare disease termed *acute ascending paralysis*, or "Landry's paralysis," in which no lesion of the cord is found after death. There has been some confusion of the two diseases, as will be mentioned in the account of this malady. The most important distinction is that in such ascending myelitis there is always much affection of sensation, and a strong tendency to trophic disturbance, while, if the patient survives, many muscles waste and present loss of faradaic irritability. In acute ascending paralysis, on the other hand, sensation is unimpaired or is merely dulled on the extremi-

ties of the limbs ; bedsores do not form, and, if the case is not fatal, there is no change in the electric irritability of any muscles.

In *meningitis* the symptoms of irritation, severe pains, muscular rigidity, &c., are prominent, while they are absent in simple myelitis, in which also the degree of fever is usually less. But in many cases the two conditions co-exist and the predominant lesion can only be decided by the order and degree of the development of the symptoms. In *meningeal hæmorrhage* there is severe pain in the back, acute irritation of the nerve-roots, and the symptoms are of very sudden onset.

Myelitis is far from rare in patients of the age and sex in which hysteria especially prevails, and many cases are mistaken for *hysterical paraplegia*. The converse error is very rare. The mistake occurs especially in cases of transverse dorsal myelitis, in which there is no wasting of the muscles of the legs. The gradual development of considerable excess of myotatic irritability is a symptom of great diagnostic value, and when this increases to characteristic extensor spasm there should be no room for doubt. Among other conclusive symptoms of organic disease, a girdle pain, and incontinence of urine or fæces, are of especial value. Trophic changes in the skin often sometimes decide, even alone, the nature of the case.

PROGNOSIS.—The danger to life in myelitis depends upon the position of the disease. It is less when the mischief is confined to the dorsal region, greater when the lumbar enlargement is affected, on account of the greater liability to trophic disturbances, which so often cause death. On this account the prognosis is worse in disseminated than in simple transverse myelitis, because some of the scattered foci are so often situated in the lumbar enlargement. The risk to life is greatest, however, when the disease is situated in the cervical enlargement, on account of the danger of fatal interference with the muscles of respiration. The prognosis is therefore rendered worse by indications of a tendency to extension, especially to extension upwards. The prospect of recovery of power is influenced by the degree of damage, as shown (1) by the degree of the symptoms, being less when sensation is lost than when there is only motor paralysis ; (2) by their vertical extent. Intense general disturbance, high fever and prostration, render worse both the immediate and the ultimate prognosis. Bedsores, if forming early, within the first month, are an unfavorable indication ; at a later period they have little influence on the prognosis. The occurrence of cystitis, and especially of any indication of secondary kidney disease, increases very much the gravity of the case. When the acute stage is over, and the disease has become stationary, the prognosis must be influenced by any indications of change that can be detected. As long as sensation as well as motion remains lost, no recovery can be anticipated, but if the sensibility of the skin has become normal, some ultimate recovery of motor power may be looked for. The development

of even an intensely spastic state of the legs does not preclude recovery of the power of walking, indeed the spasm often enables the patient to stand with a slighter degree of voluntary power than would suffice if the limbs were supple. If paralysis has remained absolute for six months, complete recovery cannot be hoped for, but some return of power, sufficient to enable the patient to walk, may occur after even twelve or eighteen months' absolute paraplegia.

The fact of preceding syphilis must be allowed very little influence on the prognosis, provided confidence is felt that the patient is suffering from a primary myelitis and not from any secondary consequence of a syphilitic growth.

TREATMENT.—If a case comes under observation at the earliest period, when only slight sensory disturbance and slight weakness of the legs indicate the commencing process, the question arises whether any treatment can avert the further development of the inflammation. If the symptoms are clearly due to exposure to cold, a hot bath, followed by free diaphoresis, should be employed, and seems sometimes efficacious. If considerable paralysis shows that the process of inflammation is fully developed, little can be expected from these measures, and it is better not to subject the patient to treatment that is incompatible with perfect rest. This, in all cases, is of paramount importance. Both functional excitation of the cord and movement of the spinal column should be avoided. The remarks regarding posture made in the account of the treatment of inflammation of the membranes apply also to that of the cord itself; it is most undesirable that the spine should be the lowest part of the body, and it is rather less difficult to keep the patient off the back in myelitis than in meningitis. If there is any reason to suspect hæmorrhage, or if there are indications of rapid extension of the inflammation, the prone position should be adopted for a time, if possible.

The removal of blood from the skin of the back over the affected region, by leeches or wet cupping, is an old measure, which finds some theoretical justification in the fact that the blood from the structures behind the spine passes into the same venous plexuses as the blood from the spinal cord itself. Hence this measure may conceivably have some influence on the circulation in the cord. If the patient's strength is not such as to render the abstraction of blood desirable, dry cupping may be employed, or the vessels of the skin may be dilated by hot fomentations, or a mustard plaster, or hot water bags. By stimulating the cutaneous nerves, these agents may also influence, in a reflex manner, the vessels of the cord. The application of cold to the spine, as by a spinal ice-bag, has also been recommended. Contrary as these therapeutic agents seem, it is probable that each moderates local inflammation in the same manner, by causing first contraction and then dilatation of the vessels of the inflamed part, and so lessening the tendency to stasis of the blood, on which some effects of inflammation

depend. Unless there is reason to suspect hæmorrhage, the application of warmth seems the safer of the two. At the very onset of inflammation mild counter-irritation is unquestionably useful, and even a blister may be employed, but when the process has reached a considerable degree, it is very doubtful whether counter-irritation has much influence until the acute stage is over.

In other respects the treatment of acute inflammation of the cord must be guided rather by the nature of the process than by the character of the organ in which it occurs. A nutritious but unstimulating diet, aperients, and diuretics, are desirable in all cases. It is extremely difficult to ascertain the influence of the drugs which are supposed to exert a special influence on the inflammation. Ergot was recommended by Brown-Séquard chiefly on theoretical grounds, and has been extensively employed. In rare cases it has seemed to do good. In cases of hæmorrhagic myelitis it may reasonably be given with greater confidence, or ergotin (3 to 5 gr.) may be injected beneath the skin. Belladonna has also been recommended, but its influence is doubtful. Mercury has been largely employed, given by the mouth and by inunction. The influence of mercury on the inflammation of internal organs does not seem so great as upon the inflammation of fibrous tissues and the structures that invest organs. Certainly in myelitis its effect is less distinct than it is in many cases of meningitis. Iodide of potassium seems to be even less efficacious than mercury. In cases of myelitis occurring in syphilitic subjects the treatment for syphilis seems to have but little influence on the morbid process. It is true that such treatment is rarely adopted at the very onset of acute myelitis, but after the disease is developed energetic treatment does not seem to modify the subsequent course of the disease. This is true also of the subacute disseminated myelitis which often occurs in the subjects of syphilis. I have known this form, concurring with syphilitic disease of the cerebral arteries, to develop and run its course to a fatal termination in spite of continuous antisiphilitic treatment. It has already been pointed out that no form of acute myelitis in syphilitic subjects presents any special histological characters. Whatever be the relation of non-specific morbid processes to syphilis, the influence of antisiphilitic treatment seems to be for the most part confined to lesions which present a specific character. Hence, in cases of myelitis in syphilitic subjects, while it may be desirable to treat the patient in accordance with the constitutional state, much effect on the morbid process must not be anticipated from the special treatment. The conclusion is that, as stated at the outset, in acute myelitis our chief reliance must be on the general measures suggested by the process of inflammation rather than on special agents. At the same time it may be thought well to use the latter in order to employ all possible means which afford even a chance of lessening a process, the most trifling diminution in which may have a profound influence on the ultimate condition of the patient.

In the general management of a case of myelitis, two points are of extreme importance. One is to avoid, by extreme cleanliness and care, the exciting causes of bedsores. The skin should be most carefully watched, and any indication of deleterious pressure met by a change of position or an alteration of the mode of supporting the part. Cotton wool is very useful for this purpose. If there is a marked tendency to trophic changes the patient should be placed on a water bed. When there is incontinence of urine, the difficulty of avoiding irritation of the skin is greatly increased. For males, a bed urinal is sometimes useful, but often it causes irritation and even sloughing of the prepuce, and then does more harm than good. A quantity of boracic absorbent cotton wool, changed as often as it becomes saturated, is one of the best means of meeting this difficulty. It must be remembered that the prevention of bedsores is the prevention of one common cause of death. Should offensive sores unfortunately form, a quantity of picked oakum, placed outside the immediate dressing, is a cheap and most effective means of preserving the air of the room from the fœtor of the sores, and therefore of increasing the comfort and wellbeing of the patient and his attendants. The second point in management is the treatment of retention of urine. If there is either simple retention or overflow incontinence, the bladder must be regularly emptied by the catheter and great care exercised to prevent the introduction of contaminating germs. If the bladder is left full and the urine allowed to dribble away, inflammation is sure to be set up, and probably also pyelo-nephritis. If cystitis occurs, antiseptic washes must be used to lessen, as far as possible, the decomposition of the urine.

When the disease of the cord has become stationary, the patient may be allowed to move and a more tonic treatment may be adopted. Iron, quinine, or arsenic may be given. Strychnia must be given only in very small doses if there is any excess of reflex action. Occasional counter-irritation may be employed, and frequently if any improvement seems to result. The limbs may be rubbed, and any muscular wasting treated with electricity. It is not desirable to use electricity as a therapeutic agent while the cord disease is in an acutely active stage. There is no evidence that the application of electricity to the spinal column has any influence on the process of recovery of the cord. Its chief value is to maintain, as far as possible, the nutrition of any muscles of which the nerves have undergone degeneration. In cases of dorsal myelitis, in which the legs are well nourished, and the reflex action is excessive, it is better not to apply any form of electricity. The unavoidable stimulation of the sensory nerves tends to increase the reflex over-action. Careful attention should, in all cases, be paid to the position of the limbs, during the stage of helplessness, so as to avoid as far as possible the development of contractions. For the condition of active spasm which often develops after severe myelitis, not much can be done; such special

treatment as can be adopted is described in the chapter on "Primary Spastic Paraplegia."

ABSCESS OF THE SPINAL CORD.

Simple inflammation of the spinal cord scarcely ever goes on to the formation of pus. In polio-myelitis, leucocytes may accumulate at certain points of the grey matter so densely as to constitute microscopic collections of pus. In very rare cases of this kind such minute abscesses have been sufficiently large to be visible to the naked eye. Pus only forms in the substance of the cord in considerable quantity in cases of purulent meningitis. In most instances the purulent meningitis has been of septic origin, rarely traumatic. In the former case, suppuration within the brain may coincide with that in the spinal cord. The latter may be very extensive. The symptoms are those of an acute irritative spinal lesion, but they are often lost in those of the purulent meningitis which precedes the disease of the cord itself. In the diagnosis of the condition, the presence of a cause of septic suppuration is the most important element in the determination of the nature of the acute spinal symptoms. A good example of the disease is a case recorded by Nothnagel. A patient suffering from cough and most offensive expectoration was suddenly seized with severe pains on both sides of the abdomen, attended by a sense of constriction and quickly followed by paralysis of the bladder and of the legs, with loss of sensation and of reflex action. An abscess of the cord was diagnosed. After death there was extensive purulent spinal meningitis, and the dorsal and lumbar cord contained an extensive collection of gangrenous offensive pus, which seemed to occupy the central part of the cord, from the cervical enlargement downwards. Some abscesses were found also in the white substance of the brain.

EMBOLISM OF THE SPINAL CORD.

The occurrence of embolism in the spinal cord has not yet been proved, but a few cases have been recorded which suggest the possibility that the process has been the exciting cause of an acute myelitis in very rare instances. In a young man with mitral regurgitation, considerable weakness of the right leg came on suddenly, with transient spasm. The onset indicated an acute lesion in the cord, which might well have been the embolic obstruction of a small vessel. In a case recorded by Weiss, a boy, aged sixteen, with chronic mitral disease, was suddenly seized with complete paraplegia, followed by bedsores, &c. He died four months after the onset, and the lumbar enlargement was found completely softened, with old coagula in the arteries. There was embolism of the kidneys and spleen, and the cortex of each cerebral hemisphere presented small foci of softening. Such cases justify a suspicion that embolism may be the cause of a sudden lesion of the cord in a patient in whom a source of embolism exists and the process has occurred in other organs.

CHRONIC MYELITIS.

The spinal cord is occasionally the seat of chronic inflammation, which develops slowly in the course of a few or many months. The lesions, which can be regarded with most accuracy as of this nature, resemble acute myelitis in seat and distribution, and intermediate cases connect them with the analogous subacute forms. They differ from acute inflammation both in the longer time occupied in their development, and in the absence of the considerable vascular disturbance which forms part of the acute process. Such chronic myelitis may be focal, disseminated, or diffuse. In the former case it may involve the whole thickness of the cord at a certain level, *chronic transverse myelitis*, or only part of it, sometimes one half, occasionally for a considerable vertical extent. The *chronic disseminated myelitis* may resemble the corresponding subacute form in distribution, many points of inflammation being scattered through a certain region of the cord, or it may affect almost all parts of the cord, and then ultimately become diffuse by the union of the areas of disease.

The term "chronic myelitis" has, however, been used in a much wider sense than that here indicated. There has been a tendency in recent pathology to extend the conception of inflammation so as to include all morbid processes which do not consist in an actual new growth. The extension is chiefly based on the difficulty of drawing a dividing line between inflammation and other processes. But it is often useful and even necessary to distinguish where we cannot divide, and it may be doubted whether any clearer conception of pathological processes has been obtained by a generalisation which is hardly more than the forced extension of an elastic name. The application of this method to the spinal cord has caused the term "chronic myelitis" to be applied to all chronic diseases other than tumours. Thus the chronic lesions that are limited to certain structures of common function, and probably begin by an atrophy of the nerve-elements ("system-diseases") have been described as "chronic parenchymatous myelitis" (Hallopeau and others). Such processes have so little in common with the forms of chronic inflammation above mentioned that it is more convenient to follow the example of those who consider them separately, as a class of "degenerative diseases," in spite of the fact that, in rare cases, the morbid process may develop with some rapidity, and indications of a secondary inflammation may sometimes be discovered. For practical purposes, it is best to base our distinctions on the salient differences of general character and course. We are not therefore precluded from recognising the points of contact between the various groups of diseases, or the presence of common elements in those that are, in the main, dissimilar. Hence we will restrict the term "chronic myelitis" to the processes already indicated as those to which it is strictly applicable.

The irregular distribution of the lesion in "disseminated" or "insular

sclerosis" may seem a special justification for including it, as many have done, under "chronic myelitis," but it presents so many peculiarities that it also is most conveniently described apart.

ETIOLOGY.—In the causation of chronic myelitis, an inherited neuropathic tendency can sometimes, although rarely, be traced. The disease is most common in the male sex and in early and middle adult life, but it occurs occasionally in old age. Many cases are met with in the subjects of constitutional syphilis, so that a causal influence of syphilis is highly probable. In most instances the primary syphilis was contracted many years before the myelitis came on. Various conditions which lessen constitutional strength predispose to the disease. The exciting causes of acute myelitis seem also capable of giving rise to the chronic form, usually when acting for a considerable time, or frequently repeated. Thus while a single intense exposure to cold may produce acute myelitis, frequent habitual exposure may cause the chronic form. Repeated over-exertion has also seemed effective in some cases. Sexual excess has been thought to be a cause, but on less clear evidence. Chronic alcoholism certainly sometimes excites chronic myelitis, usually in association with chronic meningitis; the inflammation that results from this cause is often more rapid in onset than most other forms and sometimes deserves the name subacute rather than chronic. Lead-poisoning is said to be also an occasional cause. Chronic myelitis may be secondary to other processes, as, for instance, to chronic meningitis. The inflammation that results from pressure on the cord may be chronic as well as acute. Lastly, acute inflammation may pass into a chronic stage, in which the damage to the cord not only persists but slowly increases.

PATHOLOGICAL ANATOMY.—The morbid appearances vary much in the different forms. In most cases the disease is visible to the naked eye by a grey discolouration of the white substance, from which the grey matter may be indistinguishable. The tint may be visible on the surface, even through the pia mater, which may be thickened at the spot. The consistence of the affected area may be lessened or increased; when diminished, small cavities sometimes exist in it. Rarely there is a slight increase in the size of the affected part. In some recorded cases in which there was a great increase in size, it is probable that an infiltrating glioma has been mistaken for chronic inflammation. In many cases, especially when the affected part is indurated, it is also shrunk, and the shape of the cord may be changed, as in Figs. 95, B, in which almost the whole of one half of the cord is intensely damaged. When there is one considerable focus of disease there are often other slighter foci in the neighbourhood. In what is called the diffuse form, areas of disease may be scattered through almost the whole extent of the cord. It begins at many separate places, and so is, strictly speaking, disseminated, but the areas of inflammation

may blend, so that, in the ultimate distribution, it is a diffuse inflammation. In the secondary form, the inflammation is usually confined to the neighbourhood of the morbid process which causes it.

FIG. 94.

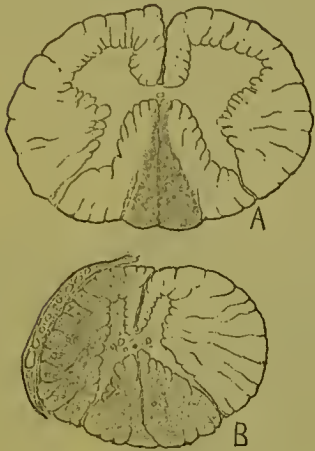


FIG. 95.

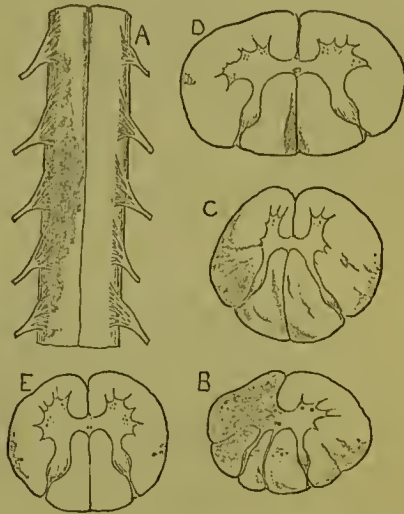


FIG. 94.—Chronic myelitis. (After Charcot and Gombault.) B, section at the level of the third dorsal, shows both the posterior column and whole of left half inflamed, except the inner part of the anterior column. The affected part was grey and uniform in aspect, vascular, and firmer than normal. Pia mater and arachnoid thickened. A, section from cervical enlargement, showing secondary degeneration in the post. med. columns. Similar foci existed in the pons, &c.*

FIG. 95.—Chronic sclerotic myelitis. (After Troisier.) A, posterior view of the affected part, lower dorsal region. The diseased areas were grey in aspect and are indicated by shading. B, middle of lesion, showing extensive damage; C, $2\frac{1}{2}$ cm. higher up, slighter damage; D, cervical region ascending degeneration, post. med. cols., and a spot of disease in lateral column (probably part of the ascending ant.-lat. tract, secondarily degenerated). E, lower part of lesion. (See next figure).†

That which is secondary to meningitis is most intense in the peripheral part of the cord, sometimes, however, extending deeply, especially when the cause of the meningitis is one, such as alcoholism, capable also of giving rise to myelitis. In this condition almost the whole thickness of the cord may be involved (cf. A and B, Fig. 97). Now and then, when there is no primary meningitis, the superficial layers of the cord

* The patient had had syphilis twenty-one years before. The spinal symptoms slowly developed during fifteen months and consisted in paralysis with hyperæsthesia on the left side, with loss of sensibility without paralysis in the right, and a band of anæsthesia to pain around the thorax at the level of the lesion, but the mental dulness of the patient prevented accurate observation during the last few months of her life. (Charcot and Gombault, 'Arch. de Phys.,' 1873, vol. v, p. 143.)

† The patient was a woman forty years of age. The symptoms slowly developed during the three years before death, and consisted of weakness with rigidity in the left leg, and impaired sensibility in the right, first of the temperature sense and afterwards of touch. No observations were made on sensibility during the last fifteen months of the patient's life, during which the lesion was slowly progressing. (Troisier, 'Arch. de Physiologie,' 1873, vol. v, p. 716, Case 2.)

are chiefly damaged, a form that has been termed *chronic annular myelitis* or *annular sclerosis* because the inflammation extends like a ring around the cord.

The microscope shows that the inflammation is essentially interstitial. The affected areas stain deeply with carmine in consequence of the increase in the connective tissue. The parts most altered may be occupied by a dense fibrous reticulum, in which few nerve-elements can be discovered, as in Fig. 96. In less affected parts of the white columns there is an irregular increase of the interstitial tissue, which has in part a fibrillated aspect, in part the appearance of an amorphous substance, studded with nuclei (Fig. 96, upper small



FIG. 96.—Chronic sclerotic myelitis, same case as Fig. 95; section near B, reversed. Almost the whole left half of the cord (right in the figure) is changed into a dense mass of shrunken connective tissue, and the right half is being invaded in many parts by the same process. The upper small figure is from the posterior column, showing a thickened vessel surrounded by nucleated tissue, among which lie nerve-fibres, many of them much smaller than normal and some with swollen axis-cylinders. The lower small figure is from the right anterior column (left in fig.), the pia mater is thickened and contains many nuclear elements; from it thick trabeculae extend into the white substance and enclose spaces, from many of which the nerve-fibres have perished.*

figure), while in other parts there may be many nucleated cells, oval, fusiform, or stellate. The large stellate "spider cells" are sometimes very conspicuous. The nerve-fibres waste before the interstitial process. Sometimes they at first undergo the change mentioned in the account of acute myelitis, by which the myelin stains more deeply than normal; afterwards the white substance becomes narrowed, and the axis-cylinder irregularly swollen, and ultimately many or all the fibres disappear from the affected area. Here and there the destruction

* From a section by Prof. Pierret, kindly lent me by Dr. Dreschfeld.

of fibres may be out of proportion to the interstitial growth, so that thickened and granular trabeculæ enclose empty spaces. This is seen in the lower small illustration in Fig. 96, which also shows the thickening of the pia mater over the affected area. In the recent state, products of degeneration, granule corpuscles, &c., may be found abundantly near the altered regions. The walls of the vessels are thickened, and sometimes the lumen of small arteries may be obliterated. Around the arteries the increase of interstitial tissue is often



FIG. 97.—Chronic meningo-myelitis, due to alcoholism. Syringo-myelia. A, lowest part of cervical enlargement; pia mater thickened, and from it tracts of tissue extend into the cord. The greater part of the section is the seat of a diffuse myelitis, in which the nerve-fibres could be seen with difficulty, and were separated by inflammatory products. The grey matter was also affected, and the nerve-cells much changed. The clear spaces in the right ant. cornu appeared occupied by products of degeneration. Central canal greatly dilated and surrounded by new growth, which in B (lumbar section) has obliterated the canal and thus caused its dilatation above. In this section the myelitis is chiefly peripheral and the grey matter has, for the most part, escaped.

most intense, and it seems to spread thence into the adjacent structures. Undue significance has been attached to this condition in old patients with arterial degeneration; it has been regarded as evidence

that arterial disease sets up the myelitis (Demange), but it is a common appearance in all forms of chronic myelitis and also in the later stages of acute inflammation. In the grey substance there is a similar increase of connective-tissue elements, so that the altered part takes a deeper stain with carmine than the normal grey substance. The nerve-elements become atrophied, the ganglion cells, at first swollen and globular, afterwards shrink, until ultimately they may be reduced to small angular bodies, and they even may disappear. The nuclear corpuscles about the central canal are often increased in quantity, and the canal obliterated, but this condition is so common in otherwise normal cords, that no importance can be attached to it. A considerable increase of tissue about the central canal, closing it below and causing it to be dilated above (Fig. 97), "syringo-myelia," has been ascribed to chronic inflammation around the canal, but the new tissue is probably of the nature of a growth, and it is doubtful whether the condition results from simple inflammation.

In chronic meningo-myelitis (Fig. 97) the pia mater is greatly thickened, and dense tracts of tissue may pass from it into the superficial layers of white substance. From these a slighter interstitial overgrowth extends in various directions. As already mentioned, this change may be accompanied by a deeper and sometimes a more diffuse inflammation, as in A, Fig. 97, in which the affection of the cord is greater and that of the membranes less than in B. The chronic myelitis that occurs in syphilitic subjects may present no specific characters. In some cases, however, the cell-formation has been abundant and has been associated with a tendency to fatty degeneration in the older portions of the lesion. The condition is, however, clearly one of chronic inflammation, and not of a syphilitic growth.*

The less chronic the inflammation, the larger is the proportion of cells and nuclei in the interstitial tissue; the more chronic, the greater the fibrillation it presents, and the firmer the consistence of the altered part. The very chronic indurating forms are sometimes called "sclerotic myelitis," or "focal sclerosis of the cord." From the various areas of inflammation secondary degenerations proceed, ascending or descending, and these may complicate the aspect of the disease at higher or lower levels.

SYMPTOMS.—The symptoms of chronic myelitis vary much according to the character of the morbid process. Different cases may manifest almost every symptom that can be produced by disease of the spinal cord, acute spasm alone excepted. Severe pain, however, is not common. There may be various motor and sensory paralyses, contractures, and even muscular atrophy. The common feature of all cases is the slow, gradual, and often successive development of the symptoms. In one of the most common forms, focal myelitis of the

* See Moxon, 'Guy's Hosp. Reports,' 1871, and Charcot and Gombault, 'Arch. de Physiologie,' 1873.

dorsal region, partial or transverse, the symptoms are paraplegic. They are the same if there is a single area of disease or if several foci develop near together. Motor power is usually impaired far more than sensibility. Subjective sensations are often prominent and early symptoms, whether there is anæsthesia or not,—tingling, “pins and needles” in the legs, a sensation as if fur or wool covered the skin, sometimes with dull pain in the legs and back, especially after exertion, and commonly also a well-marked girdle-pain at the level of the lesion. The onset of the motor weakness is very gradual; the patient gets tired sooner than before, and actual weakness gradually forces itself on the patient’s notice. Months, sometimes years, pass before the power of walking is considerably impaired. As the legs get weak, excess of reflex action usually develops, with the myotatic excess that indicates a descending degeneration of the motor fibres. The knee-jerk becomes increased and a foot-clonus can not only be obtained, but occurs from time to time when the legs are in certain positions, and a tendency to spasm gradually develops. The sphincters usually, but not always, share the impairment of voluntary power. Sensation may be unaffected, but more often some loss is found, by a careful examination, to touch, or pain, or temperature, and sometimes the impairment is considerable in degree. When there are several foci of disease, some functions may be much more impaired than others. Thus the usual rule may be reversed, and sensibility may be more affected than motion. The symptoms may come on simultaneously in the two legs, or one may be affected before the other, and occasionally the paralysis reaches a high degree in one leg while the other still possesses fair strength. This is the case when one half of the cord is affected in considerable degree, as in the lesion shown in Fig. 96. There may then even be a crossed paralysis of motion and sensation.

If chronic myelitis develops in the cervical and lumbar enlargement, the symptoms are often very irregular in distribution, and muscular wasting is usually added to the other symptoms. This occurs in the widespread diffuse form. In this, irregular muscular wasting may be the most prominent symptom, but it is always combined with other symptoms, palsy and anæsthesia, also irregular in extent and seat. The muscular atrophy may be attended by a slow failure of electrical irritability to both currents, but often some voltaic irritability in the muscular fibres persists longer than the faradaic irritability of the nerve-fibres, and sometimes there is a well-marked degenerative reaction. The symptoms may commence in the arms or in the legs, but they ultimately become general in most cases, and, according to the place of their commencement, may seem to have an ascending or descending course. The cases were described by Duchenne under the name of “*general spinal paralysis*.” In rare cases of this character, in which the disease is limited to the white substance of the cord, there may be little or no muscular wasting, but extensive paralysis and contracture, with increased myotatic irritability. Occasionally

the disease begins in the dorsal region, and after a time, perhaps years, it spreads to the enlargements.

The course of chronic myelitis is usually slow and progressive. The symptoms often increase very gradually and may only attain a considerable degree at the end of two or three years. At any stage the progress of the disease may undergo arrest. The chronic course may be varied by occasional more rapid increase of the symptoms, due to subacute or even acute processes in the diseased part. Every degree of chronicity is met with, and subchronic cases, in which the symptoms develop in the course of a few months, effect a gradation to the acute and subacute forms of myelitis. The duration of the disease varies from six months to twenty years or more. Thus in one case the symptoms slowly increased during about seven years, and then became stationary, and the patient died twenty-three years after the onset.

DIAGNOSIS.—The diagnosis of chronic myelitis rests on the slow development of symptoms indicating a random process in the spinal cord, *i.e.* a process which damages irregularly structures of various functions and thus cannot be looked on as a “system-disease.” The maladies from which chronic myelitis has to be distinguished differ according to the seat and character of the inflammation. Dorsal transverse or focal myelitis may be confounded with compression of the cord and with primary spastic paraplegia. The distinction from compression rests on the absence of a cause of compression, such as bone disease, and the absence also of the signs of a morbid process outside the cord, preceding those of damage to the cord itself. Such signs are chiefly the irritation of nerve-roots; in myelitis such irritation is never intense and is often absent, whereas it is usually a prominent symptom in all cases of compression. A tumour within the spinal cord also causes, in most cases, more irritation of the nerve-roots than does chronic myelitis. If myelitis involves one half of the cord much more than the other, the symptoms may closely resemble those of an intramedullary growth. But a tumour never causes considerable damage to one half of the cord without interfering considerably with the functions of the other half, and the symptoms in chronic myelitis may be strictly unilateral. The distinction from primary spastic paraplegia, the so-called “primary lateral sclerosis,” is often one of great difficulty. The motor state of the legs may be identical in the two diseases; in each there is the same extensor spasm, and in each there is a slow, gradual, and apparently simultaneous onset of the weakness and spasm. The distinction is that in primary spastic paraplegia the symptoms are purely motor; there is no indication that the lesion extends beyond the motor structures. In chronic myelitis, on the other hand, there is such indication, either by the impairment of sensation, or by the presence of a girdle-pain. This distinction is sufficient in the majority of cases. But, as we shall see when we consider primary spastic paraplegia, the pathological distinction

between the two diseases is not well defined, is indeed less definite than is, in the majority of cases, the clinical distinction.

The diffuse form of chronic myelitis has to be distinguished chiefly from pachymeningitis and degenerative muscular atrophy. In pachymeningitis, the muscular wasting may be similar, but anæsthesia is usually much more considerable in range and in degree, and there is often much more pain in the back. If there are similar symptoms in both arms and legs, myelitis is far more probable than pachymeningitis, since chronic inflammation of the membranes is less extensive than that of the cord. From progressive muscular atrophy the chief difference is in the random distribution of the wasting, the absence of symmetry, and the indications of irregular damage to other structures in the cord. Sensory symptoms are rarely absent in this form of chronic myelitis, while in progressive muscular atrophy, they are limited to occasional dull rheumatoid pain.

PROGNOSIS.—Chronic myelitis is a very grave disease, on account of the intractability of the morbid process, and the persistence of damage which is slowly produced. In acute inflammation there is not only a possibility of repair, but there is a tendency to it, which is absent in the chronic process. Hence, while arrest of the disease occurs not unfrequently, considerable recovery is rare. The prospect of improvement is equally small, whatever is the seat or extent of the disease. The prognosis, however, is less unfavorable the less chronic the development of the disease, and the shorter the time the symptoms have lasted. Preceding syphilis does not materially modify the prognosis; hence the great importance of the diagnosis from syphilitic growth, in which suitable treatment has a most certain effect. The danger to life is least in focal myelitis in the dorsal region; in the diffuse form the ultimate danger to life is great, but I have seen several cases in which the morbid process was permanently arrested. In chronic myelitis, as in all other diseases of the spinal cord, the prognosis is rendered worse by the presence of any of the complications which so often terminate life.

TREATMENT.—The first and most important measure is the improvement of the general health, by rest, change of air, and tonics. All causes of physical and mental depression must, as far as possible, be removed. Over-exertion, and even fatigue, should be avoided, and the patient should be kept, as far as possible, from exposure to cold. Absolute rest for a short time is often useful at the outset of the treatment, especially when there has been a somewhat rapid development of symptoms. In cases of purely chronic course, absolute rest should not be maintained for more than ten days or a fortnight, or the patient may find it hard to regain his former muscular power. Counter-irritation at the situation of the disease is often useful, and most so in cases in which there is spinal pain or tenderness. Repeated sinapisms,

blisters, or a mild form of the actual cautery may be employed. A hot douche to the back, at a temperature of 100° to 104° F., applied daily, has been strongly recommended by Brown-Séquard. Warm brine baths, and various thermal mineral waters, have been also said to do good. A sea voyage is often of service, combining as it does the maximum of fresh air with the minimum necessity for exertion. Of tonics, quinine and iron are the most useful. Drugs have, unfortunately, but little direct influence on the morbid process. Those which most deserve a trial are arsenic, very small doses of mercury (such as $\frac{1}{24}$ th gr. of the red iodide), and iodide of iron. Energetic mercurial treatment rarely does good, even when the patient has had syphilis, and sometimes it does harm. Iodide of potassium seems to have little influence on the disease. Nitrate of silver, ergot, and phosphorus have been recommended. Strychnia is chiefly useful in cases in which there is muscular wasting. The treatment of symptoms is that suitable in primary spastic paraplegia and progressive muscular atrophy, and described in the account of those diseases.

COMPRESSION OF THE SPINAL CORD.

Compression of the spinal cord is a common consequence of various morbid processes. Inflammation is almost always excited by the pressure, and interference with function occurs, partly from the pressure and partly from the resulting myelitis. The symptoms produced have, in different cases, many characters in common, although they vary according to the character of the compression, and the acuteness of the inflammation.

CAUSES.—The morbid processes that may compress the cord are numerous. Most of them have been already described. They are as follows:—Disease of the bones of the spinal column, especially caries; growths in the spine; aneurism eroding the bones and then compressing the cord; growths in the membranes; thickening of the dura mater. These processes have usually only a small vertical extent, rarely exceeding a few inches. Occasionally their vertical extent is greater, but even then the chief pressure is usually confined to a small area.

PATHOLOGICAL ANATOMY.—The cord usually presents evidence of the compression it has endured in considerable narrowing at the spot compressed, where it may be indented, flattened, or cylindrical. Sometimes the reduction in size is extreme; for an inch or so the cord may be reduced to one third of its normal diameter, and it has even been found no thicker than a crowquill. An example of flat-

tening is shown in Fig. 98. On the other hand, there is sometimes very little narrowing visible after the removal of the cord. At the compressed part the cord is usually grey in tint; its consistence is lessened in early cases, and increased in those of long duration. The change in colour and consistence are due to the inflammation of the substance of the cord which always results from pressure, and may often be traced for some distance above and below the compressed part. When there is much compression there is always much inflammation, but considerable myelitis may occur when the amount of compression is slight. The inflammation may be chronic and slow, developing in proportion to the pressure, or it may be subacute or acute, even when the pressure is gradual. The signs of inflammation are very distinct on microscopical examination, and resemble those in other forms of myelitis. There is a general increase in the interstitial tissue; in this, at first, various cell-forms may be found, but it ultimately presents the appearance of a dense reticulum. The nerve-elements undergo degeneration, and abundant masses of myelin, granule corpuscles, and corpora amylacea are visible in the fresh state or in glycerine preparations of the hardened cord (Fig. 99, c). Many nerve-fibres persist, however, with a narrowed sheath of myelin, and it is probable that these regain the power of

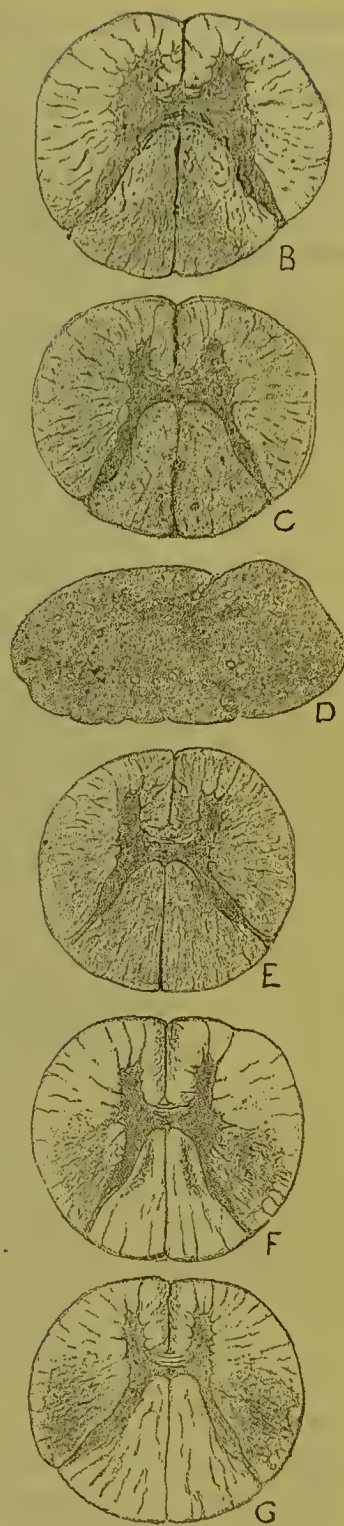


FIG. 98.—Compression of the spinal cord and pressure-myelitis, in a case of caries of the spine. D, Mid-dorsal region, near the chief point of greatest compression; the cord is narrowed from before backwards, and is uniformly damaged by inflammation, so that the grey substance can scarcely be distinguished. C, $1\frac{1}{2}$ inches higher up, shows a slighter degree of myelitis, still extending through the whole thickness of the cord. B, First dorsal; the myelitis is much slighter and the chief disease is in the posterior columns, in which ascending degeneration occupies the post.-median columns and extends outwards into the post.-external columns. E, $1\frac{1}{2}$ inches below the point of greatest pressure; inflammation still extending through all the elements of the cord. F, 2 inches lower down, shows only descending degeneration in the pyramidal tracts, anterior and lateral, and also the "comma-shaped" descending degeneration in the anterior part of the post.-external column. In G, at the lowest part of the dorsal region, the comma-shaped degeneration has ceased, and only that of the pyramidal tracts remains. (From preparations by Dr. F. G. Penrose.)

conducting, in spite of the persistence of considerable, and even dense sclerosis in the part. In extreme cases all the fibres seem to be destroyed at the point of chief compression, but there is never an actual division of the cord itself. The grey matter can scarcely be distinguished from the white, even with the microscope, and the ganglion cells become shrunken and atrophied. The walls of the small vessels are often thickened by a growth of spindle cells, more or less concentric

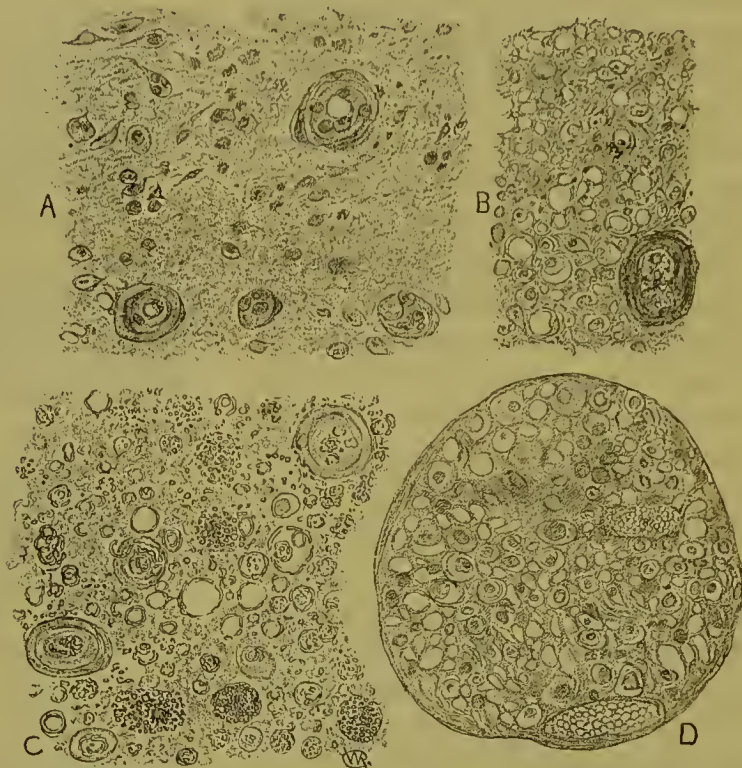


FIG. 99.—Pressure-myelitis: portions of the section D in Fig. 98 more highly magnified. A, from the grey matter, numerous angular and fusiform cells; vessels with walls thickened by a growth of cells, narrowing the cavity, which in some is obliterated. B, from the white column; thickening of the interstitial tissue, nerve-fibres in part destroyed, in part narrowed; a vessel with thickened walls. C, glycerine preparation from white substance, abundant products of degeneration of the nerve-fibres, in part aggregated into granule-masses. D, section of a nerve-root passing by compressed area; increase of interstitial tissue, many nerve-fibres narrowed, some with swollen axis-cylinders.

to the cavity, which is encroached upon and may be obliterated (Fig. 99 A, B), a process that must add to the degree of damage to the cord. The signs of inflammation gradually lessen above and below the compressed part, but often extend for some inches in each direction. Beyond its limits the usual ascending and descending degenerations may be traced (Fig. 98). The nerve-roots passing by the seat of compression suffer from the pressure, and from interstitial inflammation excited in them. They are usually grey in tint and ultimately waste, and may even be reduced to fibrous threads. The microscope shows increase of the interstitial tissue, and wasting of many nerve-fibres (Fig. 99, D), and enlargement of some axis-cylinders.

SYMPTOMS.—The effects vary much according to the degree of pressure, its rapidity, its direction, the amount and character of the inflammation produced, the amount of damage to the nerve-roots, and the position of the disease. The symptoms in most cases enable the fact of slow compression to be inferred, even when there is no indication of the cause of the compression.

The symptoms are of two classes, and it is the combination of these that is the special characteristic of the process. The first is the interference with the function of the nerve-roots at the level of the morbid process; the second is the interference with the functions of the cord itself. We may term them the “root symptoms” and the “cord symptoms.” The cord symptoms consist chiefly in impaired conduction, manifested in the parts below the lesion. The central functions of the cord (reflex action, &c.) at the level of the lesion are abolished by the pressure, but the symptoms of this abolition are often lost in those of the damage to the nerve-roots.

The first class consists of symptoms of irritation and interruption of the nerve-roots. The most constant is pain, extending along the course of the nerve-fibres, and through the area of their distribution. The seat of these pains depends on the position of the disease; they may be felt in the arms, around the thorax or abdomen, or in the legs. They are usually sharp pains, resembling neuralgia in character, sometimes attended with tender points. Occasionally, when felt in the limbs, they are referred especially to the joints. The pain may be intermitting or constant. It is sometimes increased in a remarkable degree by movement, especially when the cause of the irritation is a growth in the bones of the spine. With the pain there is usually hyperæsthesia of the skin, often intense. After a time anæsthesia develops in irregular areas, in spite of the persistence of the pain, “*anæsthesia dolorosa*.” Irritation of the motor nerve-roots may cause painful contracture of the muscles, but this is, on the whole, rare, and the chief motor symptoms are due to the interruption of the fibres—muscular weakness, gradual in onset, and accompanied by wasting. The rapidity of the atrophy varies much, and with it the electrical reaction. When slow there may be merely a progressive diminution in irritability to faradism and voltaism; when rapid there is often the degenerative reaction, and sometimes the “mixed form,” from the partial damage to the fibres that supply a muscle. Occasionally, without any actual loss of power, the patient moves the part but little, on account of the increase of the pain that movement produces,—a “pseudo-paralysis,” as it has been termed.

These local root symptoms are usually the first, and to them are added, after a time, the indications of interference with the function of the cord itself. There is weakness in the parts below the lesion, commonly developed gradually, but sometimes rapidly. Paralysis may even come on in the course of a few hours, when an acute myelitis is set up by the compression. With the weakness there is a marked

and early increase in the superficial reflex action, more constantly conspicuous than in most other diseases of the cord, and therefore of some diagnostic importance. The myotatic irritability is also increased, the foot-clonus being almost always present. Pains in the legs may occur, even when the disease is above the lumbar enlargement, usually dull and aching. There is often formication and tingling. There may be no impairment of sensibility in the parts below the lesion, even when there is complete motor palsy; in many cases, however, there is some sensory loss, complete only when the spinal lesion is very severe. Conduction of painful impressions may be delayed, sometimes for thirty or even forty seconds. When the pressure is lateral, one leg may be first and most affected, but the other side is usually soon involved, since neither the mechanical effect nor the inflammation remains limited to one side of the cord. The sphincters are often affected. The tendency to the formation of bedsores is rarely great, unless the lumbar enlargement is compressed or myelitis descends into it.

The course of the symptoms varies according to that of their cause. When an acute myelitis has been set up, improvement may occur for a time even if the cause of pressure is slowly progressive. This is true also of root symptoms when they are rapidly developed. If the compression ceases to increase, the cord may recover its conducting function, although it remains narrowed. Sensation is often regained in the legs when motion remains absent, but both motion and sensation may be regained although the narrowing of the cord persists. It has even been found no larger than a goosequill at the compressed spot, although the paraplegia had passed away. In such a case many narrowed nerve-fibres are found in the section of the compressed portion, and it is probable that many axis-cylinders persist with an envelope of myelin so narrow as to be recognised with difficulty, but which suffices for their conducting function. Under slow pressure the axis-cylinders may not suffer interruption, although the myelin wastes. Regeneration of axis-cylinders, as in the peripheral nerves, is also possible (see p. 223). In some cases, especially when a considerable myelitis has been set up, the motor path may recover and the posterior columns remain affected, so that power returns, but without co-ordination, "secondary ataxy" resulting.

The DIAGNOSIS of slow compression rests on the concurrence of the root and cord symptoms already described. Of the former, the sensory symptoms are the most characteristic. In many cases other indications of the compressing disease assist the diagnosis. These vary in character; the most frequent is considerable local tenderness of the spine. If root symptoms are absent the diagnosis is much more difficult, unless the morbid process manifests itself externally. If there is a suspicion of compression in a case of slow paraplegia, an early and considerable excess of superficial reflex action gives additional weight

to the suspicion. Even without any other symptoms to indicate compression, this is suggested by symptoms which begin on one side and gradually spread to the other.

The chief disease from which compression of the spinal cord has to be distinguished is a subacute or subchronic transverse myelitis. Besides the indications of a cause of compression already mentioned, the chief distinction depends on the presence of symptoms indicating irritation of the nerve-roots, and especially on the fact that these symptoms precede those of the lesion of the spinal cord itself. True chronic myelitis is occasionally attended by more irritation of the nerve-roots than the subacute form, but this succeeds, instead of preceding, the damage to the cord, and the course of chronic myelitis is often much slower than is that of most forms of slow compression. The distinction from a growth within the spinal cord rests chiefly on the early occurrence of impairment of the central and conducting functions of the cord, and the partial distribution, and often slower course of the symptoms. The effects of an intramedullary growth vary widely in different cases, and if there is an early irritation of root-fibres, the distinction may have to depend on the presence or absence of the signs of a compressing process.

The diagnosis of the cause of compression is often much more difficult than that of its occurrence. The facts that the patient is in the first half of life, and that he inherits a tubercular tendency, suggest caries. The absence of root symptoms is also in favour of caries or a tumour within the spinal cord. The presence of root symptoms does not, however, render caries less likely, unless the pain is extremely severe, and is greatly increased by movement, characters that should always suggest a growth in the bones of the spine. Root symptoms extending over a considerable vertical area suggest pachymeningitis. Additional details of the differential diagnosis will be found in the account of the several compressing diseases.

PROGNOSIS AND TREATMENT.—The prognosis depends on the cause, and the amenability of this to treatment. As a general rule, however, the more rapidly the cord symptoms develop, the more chance there is of improvement, because the greater share does inflammation take in their production. The treatment is that of the cause of the compression and that of myelitis.

ANTERIOR POLIO-MYELITIS. ATROPHIC SPINAL PARALYSIS.

In certain important groups of diseases of the spinal cord, wasting of the muscles is a prominent symptom. In all the lesion involves, exclusively or in part, the anterior grey cornua, and the muscular atrophy depends on the destruction of the nerve-cells, from which the

axis-cylinders of the motor nerve-fibres proceed. These fibres degenerate, and it is apparently by the agency of this degeneration that the atrophy of the muscles is produced. The occurrence of the wasting does not depend on the nature of the lesion in the grey matter; it occurs equally whether the nerve-cells are destroyed by hæmorrhage, by inflammation, acute or chronic, by slow degeneration, or by the growth of a tumour. But the rapidity with which the lesion develops influences materially the character of the symptoms. Time is required for the process of nerve degeneration, and for the effect of this on the nutrition of the muscles; one or two weeks elapse, even in the most acute cases, before muscular wasting is distinct. The nerve-cells also form part of the voluntary motor tract. Their disease at once interrupts this path. Hence, in acute cases, paralysis is rapidly developed, and the atrophy succeeds it after an interval. In chronic lesions, on the other hand, the changes in the cells and fibres progress so slowly that the weakness and wasting appear to come on together. The wasting may even seem to precede the weakness, but a careful examination will show that loss of power exists as soon as there is distinct atrophy. It is customary to distinguish the two groups by terming those in which the palsy precedes wasting, "atrophic spinal paralysis," and those in which wasting is apparently simultaneous with weakness, "spinal muscular atrophy."

The atrophic paralyses are due to an acute or subacute process in the anterior cornua. The lesion is probably, in most cases, inflammatory in character, a cornual myelitis, or polio-myelitis (Kussmaul). In some cases, which differ only in the suddenness of the onset, it is probable that the lesion is hæmorrhage and not inflammation. The chronic atrophies are due to a slow degeneration of the nerve-cells in the grey substance. By some writers it is regarded as a parenchymatous inflammation, but, in accordance with the method here adopted, the chronic disease will be classed among the spinal degenerations.

ACUTE ATROPHIC PARALYSIS; ACUTE ANTERIOR POLIO-MYELITIS;
CORNUAL MYELITIS (INFANTILE PARALYSIS; ESSENTIAL PARALYSIS OF CHILDREN).

Acute atrophic paralysis is a disease in which voluntary power is lost in the course of a few hours or days, and some of the paralysed muscles undergo rapid wasting while others recover. The onset of the palsy is often preceded or accompanied by indications of general disturbance. The muscles which waste usually remain weak, and contraction of their opponents leads to permanent distortion of the paralysed limbs. The disease is the most common form of paralysis in young children, and has hence received the name of "infantile paralysis." Before anything was known of the lesion on which it depends, it received (from Rilliet and Barthcz) the unmeaning desig-

nation of the "essential paralysis of children." The spinal lesion was first demonstrated by Prevost (1865) and soon afterwards by Lockhart Clarke, and by Charcot and Joffroy. Its constancy has been abundantly proved by numerous observations during the last ten years. The occurrence of similar symptoms in adults was noted by Vogt (1858) and Duchenne (1864).

ETIOLOGY.—The disease occurs at all ages, and although its relative frequency at different periods of life cannot yet be definitely stated, it is certainly six times more frequent in the first ten years than in all the rest of life. It is moreover especially a disease of later infancy. Of the cases under ten, three fifths occur in the first two years of life, and four fifths in the first three years. It is less frequent in the first than in the second year, but it occasionally comes on soon after birth, at the twelfth day in a case observed by Duchenne. The friends often date the disease from birth, but there is no valid evidence of its intra-uterine development. Its onset may readily be overlooked in a young child. In adults it may occur at any period, up to sixty years of age, but is most frequent between ten and thirty. Boys are somewhat more prone to suffer than girls, in the proportion of four to three, but (to judge by a series of fifty consecutive cases which I have tabulated from my own note-books) it is only under two years that a greater liability in boys can be traced. In adults, however, the disease is far more frequent in males than in females. The influence of heredity is small, but a family tendency to suffer may now and then be traced; two and even three brothers or sisters have been affected (Seeligmuller, &c.). One of my own patients had two cousins affected with the same disease. The inherited tendency may be indirect; of a patient in whom the disease came on in adult life, two brothers had suffered from hemiplegia, and an uncle from paraplegia.

The disease occurs far more frequently in summer than in winter. Sinkler, who first called attention to the fact, found that in Philadelphia four fifths of the cases commenced in the five hot months, May—September. Of my own cases, two thirds were attacked during the hottest third of the year, June—September. This relation to season is seen in adults as well as in children. The fact is the more important because cold has long been regarded as the most potent exciting cause. That exposure to cold is an occasional cause can scarcely be doubted, but it is more often traceable in adults than in children, and the extent of its influence has certainly been over-estimated. The relation to season shows that it is not the mere degree of cold which is influential, but probably the chilling of the heated body. Thus one patient, aged sixteen, when perspiring from a long ride after the hounds on a hot day in September, lay down on a sofa beneath an open window from which a draught blew on his back. Two days afterwards the first symptoms commenced. I have twice known the disease to be due to sitting on damp grass. Two or three days is the interval which

usually elapses between the exposure to cold and the onset of the symptoms.

Over-exertion has been supposed to be an occasional cause, and Lange associates the frequency of the disease in late infancy with the functional excitation of the cord in walking. Over-walking is perhaps an occasional cause in young children. A traumatic cause, such as a fall, can occasionally be traced, although the statements of friends on this point may readily mislead. "We suppose that the child must have had a fall" is a common statement, when there are no grounds for the supposition. But now and then the disease does follow a fall, at an interval of a few days, in a manner which at least suggests that the injury was its cause. Thus in one severe case, a boy eight years old was thrown over the head of a donkey, and the onset occurred five days afterwards.

Since the disease develops most frequently during the period of the first dentition, teething has, by many, been supposed to be its chief cause. But there is no proof of any relation between the two. Their coincidence in time would be significant if we knew of any mechanism by which the process of dentition could produce myelitis, or if no other conditions existed, during the period of dentition, to which the frequency of the disease might be due. But, on the one hand, we know of no such mechanism (for the theory of a reflex vaso-motor disturbance is a pure hypothesis) and, on the other hand, the period of dentition coincides with the rapid functional development of the nervous system, which succeeds its structural development in the first few months of life. Moreover, this period is often one of deterioration of health, in consequence of changes of diet and other causes. The disease occurs before and after the period of teething, and it exhibits no increase in frequency at the period of the second dentition. Of fifty cases occurring under ten, in only two did the disease occur between six and ten.

Many children are perfectly well at the time when the disease occurs. A few are in conspicuously defective health. Thus in one case the child had been rendered feeble by long-continued diarrhoea. I have only once seen the disease in the subject of inherited syphilis. On the other hand it is occasionally secondary to acute febrile diseases—scarlet fever, measles, bronchitis, pneumonia, and, in older children and adults, to typhoid fever and ague. It may develop during the height of the acute disease or during convalescence. But the frequency of this cause has been unquestionably overrated. The initial general disturbance is constantly mistaken for an independent general disease, and the opinion is often maintained after the discovery of the paralysis, which is then supposed to be of secondary origin. Thus in one case initial pyrexia, headache, and vomiting were supposed to indicate scarlet fever. Paralysis of all four limbs came on, with difficulty in swallowing, and the latter was supposed to render certain the diagnosis of scarlet fever, although there was no sore-throat or

rash. In older children and adults the disease is often thought to be secondary to rheumatic fever, in consequence of the rheumatoid character of the pains. The error is the more likely to occur if the symptoms follow exposure to cold. There is no evidence that the disease is ever secondary to articular rheumatism. Chronic alcoholism has been supposed to be a cause in adults, but it is probable that most cases supposed to be due to this cause have been instances of multiple neuritis.

SYMPTOMS.—The general characters of the symptoms have been already mentioned,—an acute onset, often with general febrile disturbance; paralysis, at first widespread, afterwards passing away except from a region in which the muscles rapidly atrophy, and in which, although partial recovery may occur, more or less weakness and wasting usually persist.

The general disturbance at the onset may be severe, slight, or absent. When present there is commonly pyrexia, with its usual accompaniments, headache, prostration, loss of appetite, restlessness. The elevation of temperature is rarely considerable, 100° or 101° , but it sometimes reaches 103° , or even, as I have seen, 104° . These symptoms occur in both children and adults. In the former there is sometimes more pronounced cerebral disturbance, convulsions, and delirium. In older children and adults transient diplopia occasionally occurs, chiefly when the disease is in the cervical region of the cord. Giddiness is sometimes complained of. The convulsions and delirium are usually ascribed to the fever, and compared to those which may occur at the onset of an acute specific disease, but it is possible that the cerebral functions are sometimes more directly deranged. These symptoms of general disturbance usually last for a few days only, sometimes for only a few hours, and they are not unfrequently absent. Before they have entirely passed away, the paralysis comes on, sometimes a few hours, sometimes a few days, after the onset of the symptoms. Thus a common history is for a child to seem ill and feverish, to be put to bed, and the next morning to be found to have lost power. As an example of more deliberate onset may be mentioned the case of a medical student who found, one day, that he saw double; on the next he felt giddy, and became feverish (100° — 102°); he slept for forty-eight hours; and on the fourth day found his right arm was weak, and atrophy of the deltoid, &c., followed. When the initial stage is prolonged, the paralysis is often not discovered for some time. The immobility of paralysis is mistaken for the inertia of prostration. When general symptoms are absent, the paralysis often comes on in the night.

In older children and adults, another common initial symptom is the rheumatoid pain already mentioned, sometimes referred to the muscles, sometimes to the limbs generally, occasionally to the course of the nerves, but never to the joints. Pain may be felt also in the

back. In character it is dull and aching, or acute and tearing or burning. It is often very severe. Instead of being initial, the pain may succeed the onset of paralysis, and occasionally continues for several weeks. There may also be tingling or formication in the limbs.

The paralysis is always rapidly developed. It varies much in its initial range. Only part of a limb may be affected, or there may be universal loss of power. In the majority of cases the paralysis is intermediate between these extremes. Two or three limbs are affected—both arms, both legs, or the legs and one arm. When all four limbs are paralysed the neck-muscles also may be weak, and even swallowing may be impaired. The other muscles supplied by the cranial nerves escape, and, as a rule, the sphincters are unaffected. To the latter, exceptions are occasionally met with, always in severe cases, and the paralysis of the sphincters, when it occurs, often lasts a long time. For instance, a child aged two and a half years woke up one morning with headache, fever, and weakness of the legs, which rapidly increased to complete paralysis. Four days later the arms also became weak, and in a day or two more the urine escaped involuntarily. The arms began to recover in six weeks, and were well in six months; both legs wasted and remained permanently paralysed; the incontinence of urine lasted for a year. A similar affection of the sphincters is occasionally met with in adults.

The paralysis usually commences in one limb and quickly spreads, reaching its maximum extent in from one to four days, and sometimes in a few hours; occasionally it occupies a week in its development. When it does not reach its height until more than a week has elapsed, the case is regarded as "subacute." As another example of a common mode of onset may be mentioned the case of a boy, aged a year and four months, who one day seemed ill, was sick, and was put to bed. The next day he could scarcely stand alone; on the following day he could neither move the legs, nor sit up in bed. In about ten days the left leg began to improve, but there was enduring paralysis and wasting of the whole of the right, and of the lower part of the left leg. Again, another child seemed ill, and was kept in bed for five days; on the third day it was noticed that the left arm was not moved so much as the right, and by the fifth day the arm was quite powerless; at the end of a fortnight improvement commenced. In the adult, the mode of onset presents nearly the same characters. A lady, twenty-five years of age, sat down for some time on wet grass. Two days later general rheumatic pains came on, very severe in the legs on the following day, and the legs were distinctly weak. In the course of the next forty-eight hours the arms also became feeble, and at the end of a week she could scarcely move her arms, and her legs were absolutely paralysed. In a fortnight improvement commenced, first in the arms and then in the feet; but the hip- and thigh-muscles remained paralysed and rapidly wasted.

Cases are sometimes seen similar in other respects to those now

under consideration, but in which the paralysis comes on suddenly, and without general disturbance. Weakness commences for instance in one arm, and spreads so quickly that in the course of a quarter of an hour there may be universal paralysis, which afterwards passes away, except in one region, where there is rapid wasting. The suddenness of the onset makes it difficult to regard the lesion as inflammation; it suggests hæmorrhage. We shall see that hæmorrhage may occur in early childhood. Hence, while we must class these with the other acute spinal atrophies, we are not justified in regarding them as cases of poliomyelitis.

The following cases are instances of such sudden onset. A girl, aged seventeen, suddenly felt tingling in the left hand. In a few minutes the whole arm was powerless, and then the right arm became paralysed. She had a strange sensation at the back of the neck, and went upstairs. As she was going up, her legs became weak, first the left and then the right. In less than half an hour from the time of the first symptom there was absolute universal paralysis, with difficulty in breathing and in swallowing. Improvement (in the opposite order to the onset) commenced the same evening and was complete in about six weeks, with the exception of the muscles of the left forearm and hand, which rapidly wasted and were permanently paralysed and atrophied. Another girl, while walking across a road, suddenly felt a "sort of shock" as if someone had given her a knock between the shoulders. She became giddy, and instantly felt tingling in both arms, especially in the right, which became weak before she had got to the other side of the road. Ultimately the arms recovered, but permanent paralysis and wasting of the intrinsic muscles of each hand were left. A similar sudden onset is also seen occasionally in children. Thus a child, aged two, was walking along, when he suddenly said he could not stand, and fell down on his knees. Both legs were powerless. Slow improvement followed in the left leg, and wasting in the right.

When the paralysis has reached its height, it remains stationary for a time, which varies from two to six weeks, and then lessens. The first improvement occurs in the parts last affected, and it gradually spreads until, usually at the end of from one to three months, all parts have recovered except those which are to be permanently affected. In these the muscles are toneless and flaccid from the first, and in two or three weeks there is distinct wasting, which rapidly increases until the shape of the limb is changed, and, in extreme cases, scarcely any of the volume of the muscle can ultimately be detected. In fat children, the appearance of the limb may be less altered, and it seems that, in some of these, an interstitial growth of fat makes up for the diminution of muscular tissue. In older children and adults the muscles are often tender to the touch during the process of wasting.

When the atrophy is distinct the muscles no longer contract to faradaism, and, if the motor nerves are tested, they also will be found to have lost irritability. The loss depends on degeneration of the

nerves. The muscles usually present the degenerative reaction in characteristic form (see p. 46). The loss of faradaic irritability is distinct, in severe cases, as early as the end of the first week and even by the fifth or sixth day. In a patient, for instance, who is universally, and apparently uniformly, paralysed, one or more groups of muscles may be found to have lost irritability, and in these we know that there will be lasting paralysis and wasting, while the other parts will recover. In a severe case, in which the muscles most affected atrophy completely, the loss of faradaic irritability may be permanent. The voltaic irritability remains, increased in degree, for two, three, four, or six months, then slowly falls, as the muscular fibres themselves degenerate, and ultimately, at the end of one or two years, it disappears. More commonly, after six or twelve months, some faradaic irritability returns. It may be slight, due to the recovery of a few fibres, insufficient in number to restore any bulk or power, but the few fibres which recover do so perfectly, so that the irritability becomes normal in degree, although the contraction which can be thus produced is very slight in amount. In other cases considerable recovery occurs, so that some power and volume are regained, although the muscles remain below the normal size. Rarely recovery is perfect. In the muscles which do not waste there is no degenerative reaction, but there may be slight diminution of excitability to each current.

The paralysis is motor only. There may be at first tingling or formication in the limbs, but there is no loss of sensation. Reflex action from the skin, however, is abolished. At first it is lost wherever there is weakness, but it returns with or soon after the recovery of power in the less affected parts. Where there is persistent paralysis it remains absent. The myotatic irritability is lost in the same or even greater degree. For instance, no knee-jerk can be obtained if the extensors of the knee are affected even in slight degree. The loss depends on the interruption of the muscle-reflex centre by the disease in the grey matter. In rare cases of severe cervical polio-myelitis, which is not limited to the grey matter but affects in less degree the lateral white columns, there is wasting of the arms, but paralysis without wasting in the legs, and in the latter the myotatic irritability may be increased above the normal, so that the foot-clonus may be obtained. Such cases are, however, very rare, and are intermediate between polio-myelitis and transverse myelitis.

A curious effect of the disease is to retard the growth of the bones in the affected limb, so that this gradually becomes shorter than its fellow, and after a time the compact tissue of the bone is less than on the other side. It is a retardation of growth and there is scarcely sufficient ground for calling it an atrophy comparable to that of the muscles. There is no evidence that any wasting of bone takes place in adults. Strange to say, Seeligmüller has observed the opposite effect in children: an actual elongation of the bones, apparently due to the fact that the growing epiphyses suffer traction instead of the

normal compression. The effect on the growth of bone is not proportioned to the muscular paralysis, and cannot therefore be regarded as the effect of the latter.

Those joints that depend for their support on tendons which pass over them become lax when the muscles are paralysed, and the articular surfaces may be no longer kept together. Thus when the deltoid is wasted, the head of the humerus may fall out of the glenoid cavity.

Bedsore are almost unknown, even in the acute stage of the disease. A slight local elevation of temperature in the most affected parts has been observed in the early stage, but subsequently the limb is constantly colder than the other. This is due, in part at least, to the want of the aid to the circulation which is, in health, supplied by the muscular action.

During the chronic stage of the disease there is great liability to the occurrence of permanent shortening of muscles, with consequent displacement of the parts to which they are attached. Grave deformities are thus produced. Unquestionably the chief mechanism by which these are produced is the shortening of muscles which are less paralysed than their opponents. The less affected muscles retain their contractility; they are no longer subject to the normal extension by their opponents, and hence gradually become shorter and less extensible. This may occur in muscles which are unaffected, and it is uncertain how far it is favoured by a slight degree of paralysis. The latter is attended with some increase of the interstitial tissue of the muscle, and the growth of this may readily fix the contracture of the proper muscular fibres. Posture often aids the production of these muscular contractions, and so also in the case of the foot does the relative shortening of the limb.

The persistent paralysis may occupy the whole or part of one or more limbs. In half the cases one leg only is permanently affected. It is less frequent for both legs or for one arm to suffer, and still rarer combinations are those of both legs and one arm, of both arms, of the arm on one side and the leg on the other, while the rarest of all is the affection of leg and arm on the same side. The limbs on the left side of the body are more often permanently affected than those on the right.

When the paralysis of a limb is incomplete, the part involved varies much in different cases, and as different parts of two or more limbs may be affected, the combinations of palsy which result are extremely varied. The grouping of adjacent muscles is sometimes distinctly that of functional association, more often it is random. In the legs the paralysis, in very severe and fortunately very rare cases, may be absolute, involving all the muscles of both limbs. Usually it is partial, and then the muscles below the knee suffer more often than those above the knee. The calf-muscles are much less frequently affected than the muscles in front of the tibia or the peronei. Hence talipes equinus is a common deformity, and its occurrence is aided by the

shortening of the limb, in consequence of which the foot has to be extended to bring the ball to the ground. Either the tibialis anticus



FIG. 100.—Old atrophic spinal paralysis. Talipes valgus in the right leg from paralysis of the anterior tibial; t. varus on the left from paralysis of the peronei.

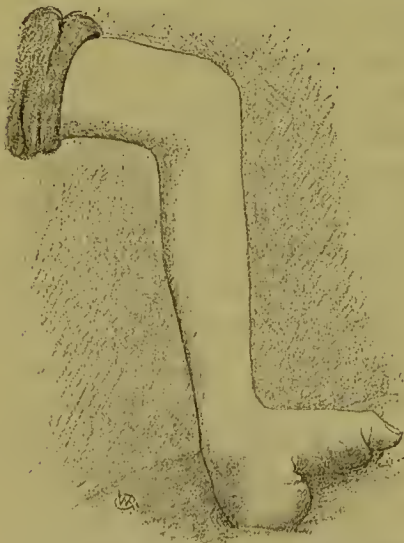


FIG. 101.—Atrophic spinal paralysis affecting chiefly the calf-muscles, with resulting contracture of the flexors of the foot causing talipes calcaneus.

or the peronei may be most affected, with the result, in the former case, of talipes valgus, in the latter of talipes varus. In the case shown in Fig. 100 the peronei have suffered most in the left leg and least in the right, producing an unsymmetrical but corresponding distortion of the feet. This affords an illustration of another fact, namely, that when there is a partial affection of both legs the paralysis scarcely ever corresponds in the two in its incidence or its degree. Much less commonly the calf-muscles suffer more than the others, and there results talipes calcaneus, as in the case shown in Fig. 101. The intrinsic muscles of the foot often suffer with those of the leg. In the thigh the extensors of the knee are affected more frequently than

the flexors, and hence flexor contraction is very common and may even cause subluxation of the joint. The flexors of the hip often suffer, with the extensors of the knee or alone. Less commonly the glutei are involved, but never alone.

In the arm almost all the muscles are sometimes affected, as in the case shown in Fig. 102, but all are never entirely paralysed. The intrinsic muscles of the hand often suffer: either the thenar muscles or the interossei may be most damaged. An instance of such paralysis of the interossei is shown in Fig. 103. The forearm muscles are frequently affected, but the supinators may escape when the other muscles are involved. The

deltoid suffers more frequently than any other single muscle of the arm. It may be paralysed alone or in association with other muscles; occasionally the deltoid, supra- and infra-spinatus, biceps, and supinators are all involved in the "upper arm type" of palsy of Erb* (see p. 77). But other muscles than these may be associated with the deltoid. In the case shown in Fig. 103 the deltoid and intrinsic muscles of the hand were wasted and no other muscles. The serratus magnus is occasionally affected, as in Fig. 102; in this case, although the left arm had suffered severely as the figure shows, on the right side only the serratus was involved. With the serratus the upper part of the pectoralis major is sometimes associated in the paralysis (as it is in normal function, see p. 26), the lower part being unaffected. It was so in this boy. The middle part of the trapezius and other scapular muscles are occasionally involved. The neck-muscles rarely suffer, but the diaphragm is occasionally paralysed. Although the intercostals and other trunk-muscles are so often weakened in the early stage, considerable permanent atrophy is very rare. Curvature of the spine is, however, often produced in consequence of the patient being allowed to sit up while the muscles are weak. It may be a lateral or an antero-posterior curve, according



FIG. 102.—Atrophic spinal paralysis, infantile form. Wasting of all the muscles of the left arm from the deltoid downwards, and of the right serratus magnus.



FIG. 103.—Atrophic spinal paralysis (adult form) affecting the interossei and thenar muscles, and also the deltoid.

as the weakness is on one or both sides. I have once seen considerable depression of the lower part of the wall of the thorax on the left side

* See a paper by Dr. Beevor, 'Med.-Chir. Trans.,' 1885.

from paralysis of the intercostal muscles at the spot, perhaps combined with hindered growth of the ribs. The muscles supplied by cranial nerves are scarcely ever affected. I have once seen a complete paralysis of one side of the face. It was associated with wasting in the limbs.

The *course* of the disease has been already sketched. There are (1) an initial stage of paralysis rapidly increasing, often accompanied with fever: the duration of this stage is from a few hours to a week; (2) a stationary period, which lasts from a week to a month; (3) a stage of "regression," during which the palsy passes away, except from certain parts in which wasting occurs: this regressive period usually occupies from one to six months; (4) a chronic stage, during which the atrophy continues, slight improvement may occur, but contractures and deformities are developed. The duration of this stage is indefinite. Improvement often occurs after three, six, or twelve months, but is then usually slight, and often more than counterbalanced by the interference with growth, and by the occurrence of deformities. Even in the slightest cases complete recovery is extremely rare. On the other hand, death from the disease is equally uncommon, and occurs almost exclusively in the early stage. It is not improbable that some children die from the initial disturbance before its nature is recognised, especially in cases attended by severe convulsions. Now and then death occurs at the end of the first week or ten days, from universal paralysis or from some cerebral complication. I have seen one example of the latter; paralysis in the arms came on upon the third day of the initial fever, reached its height on the fifth day, and then remained stationary. Faradaic irritability on the seventh day was lost in many muscles of the left arm. There was a little rigidity of the neck. On the tenth day the patient began to vomit, and complain of increased headache, and had hallucinations. On the eleventh day he became comatose and died. It is probable that other cases of the same kind occur, in which the exact nature of the spinal affection is not recognised.

Sequelæ, beyond those mentioned as part of the symptoms of the disease, are very rare. As a rule the general and nervous health of the patients and the duration of life, are uninfluenced by the local palsy. In a few cases, however, some other chronic affection of the spinal cord has come on when the subjects of infantile paralysis have reached adult life. Progressive muscular atrophy has been several times observed to start from the paralysed limb; acute and subacute polio-myelitis in adult life has also been observed, and I have twice seen the symptoms of lateral sclerosis, without atrophy, slowly developed, in one case at seventeen, in the other at twenty-eight. In the spinal cord of the subject of old infantile paralysis there seems thus to persist a disposition, slight though it be, to fresh disease, and the cases of lateral sclerosis mentioned show that the liability to disease is not limited, as has been thought, to the grey matter.*

* Compare Ballet and Dutil, 'Revue de Médecine,' January, 1884, p. 18.

PATHOLOGY.—For a long time infantile paralysis was believed to be a peripheral affection having its seat in the muscles. The opinion rested on the absence of any knowledge of the spinal changes, and on the fact of the muscular wasting. But improved methods of examining the nerve-centres have shown that changes in the spinal cord invariably exist, and, in early cases, have the characters of an acute inflammatory lesion; and we now know that muscular atrophy usually depends on central disease. Every gradation exists between the severe and the slight form of the affection, and all known facts point to the conclusion that acute atrophic infantile paralysis is invariably a disease of the spinal cord. The perfect correspondence between the infantile and adult forms, make it certain that the latter have the same origin, although pathological facts are more scanty than in the case of the former.

Observations on the changes in the spinal cord in the early stage are few. A case, recorded by Dr. D. Drummond, in all probability one of this disease, is by far the earliest on record, and is indeed the earliest possible.* A child, five years old, died after a few hours' acute illness. The spinal cord, in the region of the fourth and fifth cervical nerves, presented undue redness of the anterior grey matter. The vessels running from the surface to the cornu were distended with blood. The microscope showed distension of capillaries and minute extravasations in the grey substance, swelling of the neuroglial elements and of the ganglion cells, which were granular with indistinct processes. Another important early observation is that of Dr. Charlewood Turner,† six weeks after the onset (Fig. 104). In this, and other somewhat later cases, acute changes have been found in the anterior



FIG. 104.—Acute anterior polio-myelitis in a child $2\frac{1}{2}$ years old, six weeks after the onset. (After Charlewood Turner.) A. Section through the lowest part of the lumbar enlargement, showing a cavity visible to the naked eye, on the left side. B. Left anterior half of the cord under a low magnifying power, showing destruction of almost the whole anterior cornu. C. Portion of anterior cornu in the upper part of the lumbar enlargement. Numerous corpuscles lie in the granular protoplasm and reticulum of fine fibres. A small artery is encrusted with several layers of corpuscles and nuclei.

* 'Brain,' April, 1885.

† 'Path. Trans.,' vol. xxv, p. 203.

cornua, more advanced than in the case of Dr. Drummond. They are often widely spread in slight degree, and attain considerable intensity in one or more foci, usually in the cervical or lumbar enlargements or both. At these spots the anterior horn is softened; sometimes there is hæmorrhagic infiltration, sometimes an actual cavity (Fig. 104, A, B.). The microscope shows extravasated blood, often massed along the vessels (Fig. 104, c), and scattered through the grey matter with other cells such as are met with in myelitis. There are also granule corpuscles and other products of degeneration of the nerve-elements. These elements, and especially the motor nerve-cells, have almost entirely disappeared. Where the change is slighter in degree there is a leucocytal infiltration, a few granule-corpuscles are seen, while the nerve-cells may be structurally intact, but swollen; and granular. The morbid appearances may be confined to the anterior cornua, or may extend in slighter degree into the adjacent antero-lateral column. The posterior columns are always unaffected. In very severe cases there may be signs of slight local meningitis corresponding in position to the region chiefly affected. This region corresponds to the origin of the nerves supplying the muscles in which there is most wasting, and the anterior nerve-roots, which arise from this part, present the signs of acute degeneration. Although early observations on the morbid changes are few, a large number of cases have been examined at a later period, years after the onset. The appearances presented by these cases accord with those in the early stage, allowance being made for the difference in time. The anterior cornu at one or more places is shrunken (Fig. 105), and in part stains deeply with carmine, in consequence of its nervous structures being replaced by connective tissue, in part less deeply in consequence of "granular disintegration" of the substance. The motor nerve-cells are absent, partially or entirely. Sometimes a few shrivelled cells remain, sometimes one or more groups may be unaffected; or one or two nerve-cells of normal appearance may alone remain (see Fig. 105). The antero-lateral column is also smaller than normal and may present slight sclerotic changes, sometimes chiefly contiguous to the grey substance, sometimes extending into the pyramidal tract. In consequence of these alterations, the affected half of the cord is conspicuously smaller than the other, even to the naked eye. The consequent alteration in the shape of the cord is greatest where the damage to the grey matter is most intense. The anterior nerve-roots at the most affected part are small and grey, and the degeneration of the motor fibres may be traced down the nerve-trunks. Often, however, a few fibres present a normal appearance, although the rest have perished.

The muscular fibres, in early cases, have been found narrower than normal, and in a state of granular degeneration, with an increase in the nuclei of the sheath and of the interstitial tissue. Granules and pigment-masses accumulate between the sarcolemma sheaths. In extreme cases the degeneration proceeds to complete disappearance of

the fibres, the place of which is occupied by fibrous tracts, developed partly from the sarcolemma sheaths and partly from the interstitial

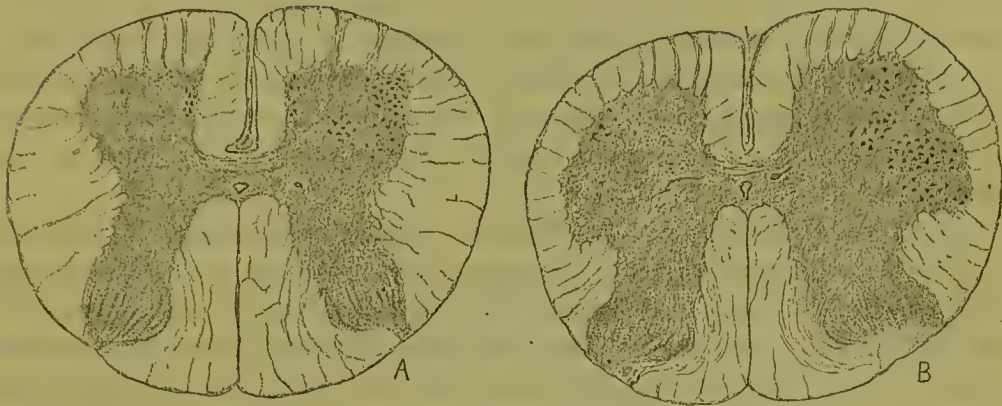


FIG. 105.—Atrophic spinal (infantile) paralysis. A. First lumbar; the left anterior cornu is smaller than the right, and its ganglion cells have disappeared with the exception of the inner group. B. Fourth lumbar; the whole left half of the cord is smaller than the other: in consequence of the diminution in size of the ant. horn. Of the ganglion cells only a few remain, belonging to the antero-lateral group. Similar changes existed throughout the lumbar enlargement.*

connective tissue. It is common to find, here and there, a fibre presenting normal appearances. Sometimes fat accumulates in the interstitial tissue, so that the reduction in the bulk of the muscle may not be so great as the actual wasting. In slighter cases, in which partial recovery takes place, some of the fibres slowly regain their normal appearance, even though they have undergone slight granular degeneration. Usually, however, many fibres perish; those which recover are smaller than normal, and the interstitial tissue remains excessive. Thus the original volume of the muscle is rarely regained.

The brain has been almost always found normal. In one case with extensive paralysis, of long duration, some atrophy was found in the motor region of the cortex (Sanders).

The symptoms of acute atrophic paralysis indicate disease of the motor nerve-cells with consequent degeneration of the motor nerve-fibres which spring from them. The pathological facts fully corroborate the conclusion, and the slighter widespread changes in the anterior cornua agree perfectly with the extensive initial palsy, which passes away. Both the character of the lesion and the mode of onset, suggest that it is inflammatory in character in the majority of cases. In rare instances, as already stated, the almost instantaneous onset suggests that the lesion is different in character, is vascular in its nature, probably hæmorrhage, although the character of the symptoms makes it probable that the seat of the lesion is the same as in those of the common

* For these sections I am indebted to Dr. H. Humphreys, of St. Leonards. The paralysis came on at one and a half years of age, two years before death, and involved most of the muscles of the left leg, those of the calf in greatest degree, so that talipes calcaneus had developed.

type. These cases have, moreover, the character of a primary hæmorrhage, not of hæmorrhagic myelitis (see p. 281). Signs of inflammation do not precede the sudden onset. Thus these cases, while they belong to the clinical group of acute atrophic paralyses, cannot at present be placed in the pathological category of polio-myelitis. They differ from the inflammatory cases in another respect, that the lesion is confined to one part of the cord, whereas in the cases that are certainly myelitic, it is frequent for different parts of the cord to be damaged.

The wide distribution of the lesion in the latter cases, and its varying degree in different parts of the grey matter, seem to preclude the theory that the mischief is set up by an initial vascular lesion, such as thrombosis. It is to be remarked, however, that when the disease is in the cervical region, paralysis of the legs does not show that the lumbar enlargement is also diseased unless there is loss of irritability in the leg-muscles or abolition of reflex action in the legs. They may be completely paralysed for a time by the damage, in the cervical region, to the adjacent lateral columns, which occurs in rare cases in which the inflammation of the grey matter is very intense.

DIAGNOSIS.—This rarely presents any difficulty except in the early stage. When the initial paralysis is passing away, and the wasting is distinct, the nature of the case is sufficiently evident, and is corroborated by the electrical reactions, by the loss of reflex action, and by the absence of any impairment of sensibility. At the onset, however, the symptoms may readily be misinterpreted. Initial febrile disturbance is naturally ascribed to some general cause until nervous symptoms manifest themselves. Even then there is risk of error. On the one hand, as we have seen, a diagnosis of a general disease is not always relinquished when paralysis appears, but the latter is regarded as secondary. On the other hand, especially in young children, the existence of paralysis is often overlooked at first, and it is supposed that the child does not move because it is prostrate. This error will not be made if it is remembered that mere prostration never causes total immobility, and *à fortiori* it does not produce local immobility. When the pyrexia ceases, and the loss of power persists and increases, the existence of paralysis is always unmistakeable. The initial general disturbance must then be regarded as part of the disease, unless there is the clearest evidence of its independent nature.

In adults, the danger of mistaking paralysis for prostration is considerably less, but, on the other hand, the general symptoms are as likely to be misinterpreted as in the case of children. The rheumatoid pains, which are so common, are usually regarded as evidence of acute rheumatism, especially when the affection follows exposure to cold. When severe rheumatoid pains are not localised in the joints, and especially when they are spontaneous, and not influenced by movement, the possibility of their spinal origin should be remembered,

and other indications of spinal mischief, such as local loss of power, tingling or formication, should be carefully watched for and receive due weight.

Of other spinal cord diseases, all chronic lesions are distinguished at once by their slow onset and gradual development. There should thus be no danger of confusing the disease with any chronic form of muscular atrophy. From other acute diseases, moreover, the distinction is only difficult in the early stage. As soon as the initial palsy begins to clear, and the muscles in one part begin to waste, the nature of the case admits of no doubt. In the early period of the paralytic stage, the disease may be suspected if the paralysis is of wide extent, and the diagnosis may be made with certainty about the end of the first week by the electrical reaction. Care is necessary in making the examination, but if properly conducted it is not attended with the slightest risk. Faradism will give all the information required, because if we find acute loss of faradaic irritability, it is certain that the characteristic alteration in voltaic irritability will follow. The isolated faradaic shocks may be employed with advantage, because the condition can be thus ascertained with much less stimulation of the sensory nerves than if the current is employed.

Acute transverse myelitis, affecting the dorsal region, causes paralysis of the legs, but, the lumbar enlargement being intact, there is no loss of electric irritability. Moreover, reflex action is not lost (except during the first day or two) as it is in polio-myelitis, and at the end of a week the myotatic irritability becomes excessive, giving rise to an increased knee-jerk and the ankle-clonus. Sensation is usually impaired at first. The sphincters are usually affected, but since they are sometimes paralysed in polio-myelitis, this indication is not absolute. If wasting occurs in these cases at a later stage, it is moderate and general, and is attended with no change in electric irritability or only with a slight diminution to both currents. But transverse myelitis, if seated in the cervical or lumbar enlargement, causes symptoms which very closely resemble those of severe cornual myelitis, since the grey matter is always implicated, and rapid wasting occurs with the same electrical reactions. Indeed, the two conditions cannot always be separated, since a severe cornual myelitis involves the adjacent lateral columns and so is, in a degree, transverse. And yet the diagnosis may often be made—by the affection of the sensory tract, and by the greater degree of affection of the lateral columns, and the occurrence of bedsores, in transverse myelitis, or by the indications of slighter disease elsewhere in cornual myelitis. Thus transverse myelitis in the cervical region causes total and long-continued paralysis of the legs, often with anæsthesia; in cervical cornual myelitis the paralysis of the legs, if it occurs, is incomplete, and soon improves, while sensation is unaffected. In cornual myelitis of the lumbar enlargement there may be slight and transient paralysis of one or both arms—an indication that the morbid process is dis-

seminated in distribution, which is absent in transverse lumbar myelitis. In extensive spinal hæmorrhage the paralysis is absolute; there is pain in the back, and affection of the sphincters, while bed-sores rapidly form, and the onset is sudden. Cornual hæmorrhage, as already stated, is distinguishable from cornual myelitis only by the suddenness of the onset. The separation of subacute from acute myelitis of the grey substance is, to a large extent, arbitrary. The symptoms are similar, but the mode of onset is less rapid and occupies more than a week. The distinction of diphtheritic paralysis and of multiple neuritis is from the subacute rather than the acute spinal atrophies, and is considered in the next section.

The diagnosis from paralysis of cerebral origin is usually easy. It has to be made chiefly when the palsy is bilateral or hemiplegic, or, in the chronic stage, when the arm is paralysed and the case simulates hemiplegia with recovery of the leg. Careful attention to the symptoms will generally prevent error. Loss of faradaic irritability never occurs in cerebral paralysis, nor does extreme muscular wasting. The skin-reflexes may be lost in each, but the muscle-reflex action (myotatic irritability) which is lost in the spinal affection, is always preserved, and often excessive, in cerebral palsy. In the latter, in children, mobile spasm and spastic inco-ordination are common in the hand, while in spinal infantile paralysis they are always absent. Slight paralysis (or spasmodic over-action) is common in the face in cerebral hemiplegia. Any cerebral symptoms which may attend the onset of polio-myelitis, convulsions, delirium, diplopia, headache, are brief in duration, whereas in cerebral lesions they may be very severe and continue for some weeks. The convulsions which attend the onset of infantile spinal paralysis are general, while in a cerebral lesion they are usually unilateral or commence unilaterally.

The exceedingly slow onset of pseudo-hypertrophic paralysis, developing gradually, as it does, with the child's growth, ought to render its confusion with polio-myelitis impossible. Cases in which the muscles are small instead of large, are sometimes confounded with acute spinal paralysis, but only by inattention to the absolute distinction of the mode of onset.

Diseases outside the nervous system which have been mistaken for infantile paralysis are chiefly those in which local pain interferes with the movement of the limbs, and the child is young. I have known, for instance, the mistake to be made in the case of hip-joint disease, necrosis of the femur, and the affection termed "scorbutic rickets," in which there is enlargement of the shafts of the long bones, extreme pain on movement, and spongy gums. In all these cases, a careful examination will show that movement is interfered with only by the pain; there is no actual paralysis, and there is no interference with reflex action. The preservation of the myotatic irritability is often of considerable assistance in these cases. It is common, for instance, for the extension of the knee to be interfered with, but the preservation

of the knee-jerk at once proves that atrophic paralysis is not the cause of the defect.

PROGNOSIS.—The danger to life, in cases in which the nature of the disease is recognised, is extremely small, but it is possible that children sometimes die from the severity of the initial general disturbance, before the development of the characteristic paralytic symptoms. In the stage of paralysis there is danger to life only when the chief disease is in the cervical region. Death may occur at the end of a week or ten days from interference with respiration; the degree of which is a measure of the danger to life. Definite cerebral symptoms are also of very grave significance. In the vast majority of cases the disease entails no immediate danger to life. But children are left with little power of resistance to other morbid influences, and occasionally succumb to some other illness, as an acute specific disease, or an attack of bronchitis, a few weeks or months after the onset of the paralysis.

As soon as the paralysis has become stationary, *i. e.* has not increased for twenty-four hours, the danger of further extension is extremely small. But the question at once arises, and is anxiously asked,—What will be the permanent condition? Will there be lasting paralysis? An answer cannot be given until the end of the first week or ten days, and then only by means of an electrical examination. Whatever muscles, at the end of that time, have lost faradaic irritability, will certainly waste and remain for a long time paralysed. On the other hand, if there is no loss of irritability at the end of ten days, but it is apparent at the end of a fortnight or three weeks, the wasting will be slighter in degree, and considerable ultimate recovery may be confidently looked for even in the most affected part. Where there is no loss of irritability, the paralysis will pass away in the course of a few weeks, or at most of a few months. Where irritability is lost tardily, there will be wasting and paralysis for several months. Where irritability is lost early the wasting will be rapid and great, the paralysis will last for one or several years, and it is unlikely that perfect recovery will take place. Without an electrical examination it is necessary to wait longer before a prognosis can be given, until distinct wasting on the one hand and improvement on the other, indicate the region in which the paralysis will persist and in which it will pass away.

In the chronic stage the prospect of ultimate recovery depends on the rate at which wasting developed, on the electrical reaction, and on the duration of the case. Where the wasting is great and has been rapid, and faradaic irritability is entirely absent, although some recovery may occur, it is not likely to be complete, and if this condition exists a year after the onset, it is improbable that more than very slight improvement will occur. On the other hand, if, at the end of one or two months, some faradaic irritability can be still detected,

although low in degree (*i. e.* elicited only by a strong current) considerable improvement is probable, and actual recovery is possible at the end of six or eight months. It is necessary, in the case of children, to remember, and warn the friends, that the growth of the most affected limb will be hindered, and that this, in the case of the leg, may render the effect of the paralysis more obtrusive by its interference with the gait, although real improvement may be taking place.

TREATMENT.—The treatment of the acute stage of the disease is essentially the same as that of myelitis, already described, and the rules and principles already stated need not be here repeated. In the initial stage it should be that of general febrile disturbance, consequent on a local inflammation. In such a disease, in which there is a natural tendency for the morbid process to cease to spread, and then to lessen in extent, the difficulty of ascertaining the actual effect of the drugs employed is extremely great, and hence the very different opinions which have been expressed regarding their influence.

When the acute stage is over and the condition of the patient is stationary, the general treatment should be tonic, iron and quinine being the most useful. The influence of strychnine is also probably beneficial, although its use is undesirable while the motor elements of the cord are the seat of an active morbid process.

Electricity has been strongly advocated, and largely used, in the treatment of this disease, and there is reason to believe that it is useful, although its influence has been much exaggerated. In no sense is it a curative agent, and there is no evidence that its application to the spine is capable of increasing the degree or accelerating the course of the recovery of the nerve-elements. Nor is it easy to obtain evidence of its influence over the muscles. If the wasting is rapid, this progresses in spite of daily and sedulous applications. Nevertheless its demonstrable effect on the muscles in causing their contraction must have an influence in the right direction upon their nutrition. Faradisation is useless, since the muscles cannot respond to it, but to the voltaic current they act readily, and their daily stimulation by its means tends to maintain their contractility. This is, it is true, of no avail if no recovery takes place in the spinal cord, but in most cases some recovery in the cord does occur, and when, after months, the nerve-elements regain the power of conducting the voluntary impulse, the muscles are in a better condition to respond to it, if they have been regularly galvanised, than if they have been left alone, a prey to unhindered degenerative processes. The influence of electricity is clearly shown by cases in which no treatment has been adopted for six or twelve months after the onset. No response may at first be obtained to the current, but after a few applications contractility returns, and is often followed by slight voluntary power. The mode of application should be to place the positive electrode on the upper part of the muscle and to stroke the negative down it. The application can be

made perfectly well by an intelligent nurse, since the diffusion of the voltaic current is so great that there is not the same necessity for the exact localisation of the electrodes, as in the use of faradaism. Large well-wetted sponges should be used. The current should be no stronger than will cause distinct contraction in the muscle. In young children the application sometimes occasions considerable emotional disturbance. This may often be avoided by commencing with a very weak current. It is better, indeed, to commence with no current at all, applying the sponges in the way directed, so that the child may be familiar with them, and cease to fear them. Then two or three cells may be used, and the strength gradually increased day by day. In this way a current strong enough to cause contraction will often be tolerated. If, however, in spite of these precautions, this strength cannot be employed without distressing and disturbing the child, the attempt to obtain muscular contraction should not be persevered in, but only such a current employed as does not disturb the child. The influence of the current, if slighter, is still in the right direction, and the slightly greater influence of the stronger current is more than counterbalanced by the harm caused by daily severe emotional distress. The electrical treatment may be commenced at the end of the third or fourth week after the onset. It should not be used earlier, lest it excite increased disturbance in the spinal cord. The application need only be made to those muscles in which faradaic irritability is lowered or lost. Other muscles will either recover perfectly without its aid, or (as in the case of paralysis of the legs from implication of the lateral columns in the cervical region) will be uninfluenced by electricity.

Another measure which should be employed is systematic rubbing of the limbs. This stimulates the circulation, which is always defective, as the blue, cold surface shows. It no doubt also increases the movement of the fluids in the tissues outside the vessels, and so probably increases the interchange of material. The muscles should be daily rubbed, and kneaded, and gently pinched. The rubbing should be especially upwards, so as to expedite the movement of the blood in the veins. No liniment is needed; the uncovered hand answers best. Cod-liver oil is sometimes rubbed in. There is no other objection to this than that it is apt to distract the attention of the rubber from the real object, and therefore from the proper method of the rubbing. But the oil does not reach the deeper structures of the limb sooner or in greater degree than if taken into the stomach. Great care should be taken, at all times, to keep the affected limbs as warm as possible.

In all cases in which the back-muscles are weak even in slight degree, bronchial catarrh must be guarded against with great care. In all such cases the muscles of respiration are weakened, and although normal breathing may not be impaired, the diminution in strength may render an acute bronchial catarrh rapidly fatal, and this even months after the onset of the paralysis.

A very important element in the management of the chronic stage is the prevention and treatment of the muscular contractions, and of the deformities to which these give rise. The contractions cannot be entirely hindered, but they may often be prevented reaching a high degree by careful attention to the position of the limbs, and by watching for and treating the earliest indication of shortening of the muscles. It is needless to discuss in detail the varied influence of posture in these cases; careful and frequent examination of the patient will show whether any deviation from normal relations is being developed, and its counteraction is chiefly a matter of common sense, and a little practical ingenuity. Of especial importance, however, are the prevention of the curvatures of the spine which are caused by allowing the patient to sit up before the back-muscles have regained the necessary power, and the prevention of the contraction of the flexors of the hip and knee-joints which occurs when the patient is allowed to lie in bed with the legs drawn up. More difficult of prevention is the contraction of the calf-muscles, often aggravated in consequence of the lessened growth of the limb. Something may be done during the process of rubbing, for the prevention of deformities. While the contracted muscles are rubbed upwards, they should be extended gently but firmly. Thus, if the calf-muscles are shortened the foot should be steadily pressed upwards while the calf is rubbed. A light Scarpa's slipper, worn at night, often aids in overcoming the contraction at the ankle-joint. For developed deformities, due to great shortening of the muscles, surgical treatment by splints or tenotomy is usually necessary.

Mechanical appliances are of unquestionable value in the treatment of these cases during the chronic stage. They tend to counteract deformities, and to supplement weak muscles. Thus they often enable a child to walk, who could not do so without their aid. Achieving this they also do more. If a child cannot attempt to walk, muscles are unused which have regained some power, and power is often increased to a remarkable extent when instrumental aid enables these muscles to be brought into active use. In all these cases, however, the instruments require frequent attention and alteration to adapt them to the changes due to growth. Much patience and perseverance are required in the management of the case through its long and tedious course.

SUBACUTE AND CHRONIC ATROPHIC SPINAL PARALYSIS
(SUBACUTE AND CHRONIC POLIO-MYELITIS).

Under this designation cases have been described in which paralysis, followed by muscular atrophy, comes on less rapidly than in the acute form, its development occupying from ten to thirty days, in the cases which are termed "subacute," and from one to six months or even more, in those which are called "chronic." The shorter the period occupied by the development of the disease, the more distinctly does weakness precede wasting, and the more marked is the degenerative reaction in the muscles. But the cases included under this name differ much in their characters, and certainly belong to several distinct forms of disease. Some are diseases of the spinal cord, others are diseases of the nerves. The spinal forms are rare. The majority of instances of the subacute form are examples of multiple neuritis. The cases of atrophic paralysis in which the onset is not acute may be placed in three classes.

(1) Cases of subacute cornual myelitis which differ from the acute form, already described, only in their slower development. They present the same initial general disturbance, and wide extent, and the same recovery, except in a limited region in which muscular wasting occurs. Most of the sufferers have been adults. Little is known of the exciting causes of this form further than that it certainly sometimes follows exposure to cold. Frequently, however, no cause has been traced. The symptoms and general history of these cases present no important difference from the acute form.

(2) Cases which differ from the type just described in the important characteristic that their course is progressive instead of being regressive. The onset is subacute or subchronic, occupying from a fortnight to several months, but instead of arrest followed by improvement, the more or less rapid onset is followed by slower gradual increase, until at last widespread chronic muscular atrophy is developed. This variety was first described by Duchenne in 1849. By some it has been regarded as merely a chronic form of the acute affection, but the difference in course makes it probable that, as Erb suggests, it is really a distinct malady. This form also is chiefly confined to adults. It occasionally results from cold, and sometimes from injury, as a fall on the back. Intemperance, and venereal excesses are also said to cause it. The widespread muscular atrophy which sometimes results from lead-poisoning is probably of the same nature.

In the subacute cases there may be slight fever at the onset. Pain in the back is common, and sometimes there is tingling or formication in the limbs. Loss of power usually commences in the legs, and steadily increases, with loss of all reflex action, but with no affection of sensibility. Muscular wasting occurs irregularly, some groups of muscles being chiefly involved. The upper limbs are affected after the lower, but in a similar manner. The muscles usually present

the degenerative reaction, loss of faradaic irritability with increased and altered voltaic irritability, but in some cases, in consequence of the unequal affection of the nerve-fibres, there is the mixed form of degenerative reaction (p. 21), faradaic irritability is preserved in the nerve, while in the muscles voltaic irritability is increased and altered. In many of the more chronic cases, however, there is a simple loss to both faradism and voltaism. The sphincters almost invariably escape. Loss of power usually precedes wasting. In some cases, the affection exhibits a persistently progressive character, muscular atrophy slowly increases and extends, until the case ultimately resembles one of progressive muscular atrophy, and the patient dies at the end of one or two years from exhaustion, or from interference with the respiratory movements. In other cases, the atrophy, after slowly progressing for many months, becomes stationary, and considerable improvement may ultimately take place. This course is seen especially in traumatic cases. Few observations on the pathological anatomy of the affection have been made. Cornil and Lépine found, in one case, at the end of four years, softening of the lower part of the spinal cord, chiefly in the anterior cornua, disappearance of the ganglion cells, increase of the connective tissue, and sclerosis of the white substance around the anterior horns. The diagnosis from the first form rests on the slow progressive onset and course of the disease, and the fact that it either does not become stationary at all, or not until after many months. Although the degenerative reaction may be present in the muscles, it is frequently absent, and this is another distinction from the acute variety. On the other hand, the fact that paralysis precedes wasting is a distinction from progressive muscular atrophy. In the most chronic form, however, this distinction fails, and cases are met with which present a gradation between the two diseases. The prognosis is grave except in traumatic cases, but is influenced by the observed rate of progress, and the presence or absence of any indications of arrest. When the result of injury, considerable improvement is not uncommon, and it is remarkable how great a degree of paralysis and atrophy may, in these cases, ultimately pass away almost completely. The treatment of the disease must be influenced by the rapidity of its onset. In the subacute stage and form it should be conducted on the same principles as that of acute cornual myelitis. In the chronic form the treatment must be the same as for progressive muscular atrophy.

(3) There is no doubt that many subacute and chronic cases, which have been described as atrophic spinal paralysis, are peripheral, not central in their nature, and are cases of multiple neuritis, the symptoms and diagnostic indications of which have been already described. The history of the subacute spinal disease has been largely written from cases of multiple neuritis.

ACUTE ASCENDING PARALYSIS.

Several diseases cause rapid palsy, which commences in the legs and ascends to the muscles of the trunk, the arms, muscles of the neck, the diaphragm and the pharynx. Such ascending paralysis may be caused by meningeal hæmorrhage, and by ascending myelitis, but it occurs also in cases in which the other symptoms of these lesions are absent, and in which the spinal cord, after death, appears healthy to both naked eye and microscopical examination.

The pathological nature of these cases is mysterious, but they cannot be classed with other recognised lesions of the spinal cord. The affection has been provisionally termed "acute ascending paralysis" or "acute general paralysis," in the absence of any evidence of its nature. It was first described by Landry in 1859, and hence is often called *Landry's paralysis*. Acute ascending paralysis is a formidable malady, most cases proving fatal in a few days.

CAUSES.—The remote etiology of the disease resembles, in general, that of acute myelitis. The disease affects males more frequently than females. It occurs chiefly between twenty and forty years of age, but has been observed, in rare cases, in older and younger persons, and even in children. Severe exposure to cold has been the exciting cause in many instances. The symptoms have occasionally come on during convalescence from some general disease, smallpox, diphtheria, typhoid fever. The affection has been ascribed to syphilis, and the symptoms have been said to pass away under antisiphilitic treatment. Difficult as it is to conceive by what mechanism this cause can be effective, many cases of the kind have been described.

SYMPTOMS.—Premonitory symptoms have been noted in a few cases, —general malaise, pains in the head and back, tingling in the extremities—for a few days or a week before the onset. The first definite symptom is usually weakness of the legs, often commencing in one and spreading to the other. The weakness increases rapidly, so that the power of standing is lost, sometimes in a few hours, sometimes at the end of two or three days, and it goes on to complete paralysis, with relaxation of the muscles. As the legs become motionless the muscles of the trunk become weak, first those of the pelvis, loins, and abdomen, then of the thorax. The weakness next invades the arms; either the upper arm muscles or those of the forearm and hand may be first attacked, and one arm is often weakened before the other. The paralysis of the arms may become absolute, like that of the legs, or some power of movement may remain. The diaphragm and neck muscles then suffer, and difficulty of swallowing comes on, sometimes with paralysis of the muscles of the palate, and often speech becomes

difficult, nasal, and indistinct. The inability to swallow may become so great that the patient has to be fed through a tube, and the paralysis may involve the muscles of articulation to such an extent that utterance may be altogether unintelligible. There is dyspnoea from the weakness of the muscles of respiration.

The tingling and analogous subjective sensory disturbance have been followed, in some cases, by hyperæsthesia of the skin, and tenderness of the muscles. There may be some blunting of sensibility in the extremities as the paralysis becomes absolute, but there is no considerable loss of sensation. A firm touch can usually be perceived anywhere; perception of painful impressions and of heat or cold is sometimes delayed. At first reflex action is lost in the affected limbs, both cutaneous reflex action and myotatic irritability. In cases rapidly fatal the loss has continued till death. In cases that have recovered, reflex action has returned, but recorded cases have presented considerable differences in this respect, and, as there is some doubt as to the nature of many non-fatal cases, there is some uncertainty as to this point. In some the myotatic irritability has soon returned and has even become excessive. In others it has remained absent, and not until all paralytic symptoms have disappeared, has the knee-jerk returned.

In spite of the early flaccidity of the muscles, if life is prolonged they present neither wasting nor change in electrical irritability. A trifling reduction in size may occur, but there is no muscular atrophy such as occurs in poliomyelitis, and even after several weeks the most careful examination fails to reveal any abnormal electrical reaction. According to observations by Remak and others, this may be regarded as characteristic of the disease. The sphincters, moreover, escape in the vast majority of cases, but not in all; there is no tendency to the occurrence of bedsores. The cerebral functions are not involved, and the state of the patient who, with unimpaired intellect, cannot express himself either by speech or gesture, is painful in the extreme.

As a rule there is no elevation of temperature, even during the rapid development of the symptoms. It is not, however, certain that there is never initial fever. In one or two cases, which were not fatal, brief pyrexia attended the onset, and in a few others febrile disturbance occurred during the subsequent course of the affection. In at least one febrile case the symptoms passed away with a rapidity and completeness that seem incompatible with an ordinary inflammation of the cord. An acute enlargement of the spleen was first noted by Wesphal, and has since been observed in several other cases, so that it is perhaps constant.

Thus the chief feature of the disease is an almost purely motor paralysis, progressive in character, with relaxation of the muscles during the acute stage of the disease. Although usually ascending in course, the arms are occasionally involved before the legs, and the bulbar symptoms have been known to precede the others.

The muscles of respiration are sometimes little affected until after the paralysis has reached the throat. Very rarely other cranial nerves have suffered in slight degree; there has been diplopia, paralysis of accommodation, or dilatation of one pupil. In one case the face was paralysed on both sides. The disease varies considerably in the rate of its progress. It may run its course and end in death in forty-eight hours. A large proportion of the fatal cases last less than a week. On the other hand, the disease may only attain its height at the end of two, three, or even four weeks. In some cases, apparently of the same character, the paralysis of the limbs has not been complete and bulbar symptoms have been absent. There may be a rapid extension of the paralysis to a certain point, and it may then cease to spread, and the limbs last and least affected may slowly regain power, the order of recovery of the muscles being the opposite to that of their invasion. When recovery has occurred, two or three months have usually elapsed before the weakness entirely disappeared, but in a few cases there has been a more rapid improvement and the patient has been well in a few weeks. It is probable, however, that some cases recorded as recoveries from this disease have been cases of myelitis, or of rapid multiple neuritis.

PATHOLOGY.—In several cases the most careful and skilled examination has failed to discover any morbid appearance in the spinal cord, nerves, or muscles. Minute hæmorrhages, met with occasionally, have probably occurred during the last moments of life. In a few cases, apparently similar to the others, slight scattered indications of inflammation have been found in the grey matter of the cord, with degenerative changes in the ganglion cells (Immermann). In addition to the enlargement of the spleen observed during life and found also after death, the abdominal glands have been found swollen. Bacteria were found in the glands by Baumgarten, but others (Wesphal, Kahler and Pick) have looked for them without success.

In the absence of any constant anatomical changes, the nature of the disease is a matter of speculation, which is the more uncertain since the clinical separation of this from other maladies is often difficult. The traces of inflammation in the grey matter, which have been found in a few cases of ascending course, have been regarded by some writers, in France especially, as an indication that the disease is really a form of poliomyelitis, inseparable from the common form of acute atrophic paralysis. But the fact that, in patients who survive, there is no wasting and there is no change in the electrical irritability of the muscles, seems opposed to this view, and the common absence of fever makes it very difficult to regard the lesion as a primary inflammation. These considerations, coupled with the frequently rapid course of the malady, have suggested to many pathologists the idea of a toxic influence acting on the cord; and this idea receives support from the discovery that acute swelling of the spleen is common, and of the lymphatic

glands not rare. Such lesions indicate a morbid blood-state. The fact that paralysis of accommodation has been observed affords some confirmation of this view, since the isolated acute paralysis of a functional centre, not anatomically separate from others, is known only as a consequence of a toxic influence. If this hypothesis is admitted as probable, the question remains—on what nerve-structures is this toxic influence exerted? It is clearly upon some part of the motor path. The absence of atrophy or altered reaction of the muscles shows that the interruption is not in the lower segment of the motor path (p. 116), while the flaccidity of the muscles and the prolonged impairment of myotatic irritability suggests that the structures on which the morbid influence acts are not far from the lower segment, are in or near the grey matter in which that segment begins. Curara abolishes the function of the termination of the lower segment of the motor path, *i.e.* of the nerve-endings in the muscles. It may be that, in this disease, some toxic influence impairs in an analogous manner the function of the termination of the upper segment, paralyses the ramification in which, as we have seen (p. 115), the pyramidal fibres must end in the grey matter, and by which they are connected with the lower segment.* It has been already pointed out (p. 144) that the nutritional stability of the termination is probably lower than that of any other part of each segment, since it is the part farthest from the nerve-cell from which the fibre proceeds, and on which its nutrition depends. The greater length of the fibres for the lumbar region may render their termination more susceptible than that of the fibres for the arm, and hence the ascending course of the paralysis. It is, moreover, readily conceivable that the effect extends in slight degree to other structures in the grey matter; those for instance, which intervene between the sensory and motor cells, and thus the loss of reflex action and myotatic irritability are explained, as well as the muscular relaxation, since, as we have seen, muscular tone must be ascribed to a reflex process. The structure affected may vary somewhat in different cases, and thus the state of reflex action may vary. The facts of diphtheritic paralysis also show that the effects of such a toxic influence may lead to secondary inflammation, and we can thus understand that indications of such inflammation in the grey matter may be occasionally met with. The bulbar symptoms come under the same explanation. The motor path through the nuclei of the medulla has the same pathological relations as that which passes through the grey matter of the spinal cord. The separate affection of nerve-structures, distinct from others only in function, is common in the phenomena of toxic actions. There are no facts to show the nature of the blood state, its causes, or alliances, except that on the one hand the frequent relation of the disease to cold suggests an analogy with rheumatic blood-states, while, on the other, its occasional relation to acute specific diseases is noteworthy. Perhaps the malady to which

* The symptoms of lathyrism suggest that such an effect is actually produced in that toxic state.

acute ascending paralysis bears the closest analogy is diphtheritic paralysis. The supposed relation of some cases to syphilis is as obscure as are most other points in the pathology of the disease.*

DIAGNOSIS.—The disease is recognised by the rapid development of ascending palsy with relaxation of the muscles, and with loss of reflex action, without fever, considerable pain, or loss of sensation, and if the patient survives, without wasting of the muscles or change in electrical irritability. The latter characteristic distinguishes the disease from acute atrophic paralysis, while the absence of pain in the back and of spasm is a distinction from meningeal hæmorrhage. The diagnosis from general ascending myelitis has been already considered (p. 231).

PROGNOSIS.—The affection is one of extreme gravity. The danger to life is in proportion to the interference with respiration and with the functions of the bulbar nerves, and also to the rapidity with which the palsy develops. But the cases which develop with comparative slowness are not devoid of danger. Even when the symptoms only reach their height at the end of three or four weeks, death may occur in the same way as in the more rapid cases. On the other hand recovery has been known, although the patient has lost all power of motion at the end of the second day. The danger is great as long as the symptoms are increasing, and only when distinct improvement can be recognised is it justifiable to anticipate recovery.

TREATMENT.—During the early stage of an attack of acute ascending paralysis, the treatment must be that suitable for acute myelitis, since, at the onset, the diagnosis between the two diseases can never be certain. A warm bath, or still better, a vapour bath, should be given if the symptoms followed exposure to cold. It should be followed by counter-irritation over the spine. A long, narrow mustard plaster may be applied to the entire length of the spinal cord. More energetic counter-irritation, even the actual cautery, has been recommended. The little that is known of the pathology of the disease suggests that the dorsal position may be less injurious than in myelitis; but until the nature of the case is clear, the patient should be kept on the side if this does not involve frequent changes of posture. The body should be kept in as perfect rest as possible. In very few cases have drugs appeared to exert any influence on the course of the disease, and the malady is so rare that experience accumulates slowly. Salicylate of soda seems to deserve a trial in cases that follow exposure to cold. Ergotin has been given, and one case in which it was used deserves special mention. The patient was a man aged fifty-seven, who, a week after exposure to cold and wet, complained of a feeling of weight and weakness in

* If a blood-state arises in the course of syphilis, capable of abolishing the function of certain nerve-structures, the fact is of great interest in connection with the relation to syphilis of degenerations of the spinal cord.

the legs; the temperature rose to 103°; the loss of power gradually became complete in the legs and spread to the arms, without loss of sensation. At the end of the second day there was difficulty in swallowing, in articulation, and in breathing, and death seemed near. Ergotin was given every hour, and during the night the patient took twenty grains. In the morning the bulbar symptoms were better, the arms stronger, and there was a trace of motor power in the legs. The patient rapidly improved, and at the end of a week was well.* If swallowing becomes difficult, care must be taken to administer a sufficient amount of nourishment, either by the rectum, or by the nasal tube.

PARALYSIS FROM LESSENERD ATMOSPHERIC PRESSURE: DIVERS' PARALYSIS.

Divers, and those who work in caissons, at such a depth beneath water that they are exposed to considerable pressure, sometimes become paraplegic soon after their return to the surface. Apoplectic attacks and hemiplegia also sometimes occur, but paralysis of the legs is by far the most common effect, and it is, therefore, clear that the spinal cord suffers in greater degree than any other part of the nervous system. Miners have been said to suffer also, but this is doubtful, because a pressure equal to at least an additional atmosphere seems necessary for the production of the symptoms. Most of the subjects of the disease have worked at a depth of from forty to ninety feet below the surface of water. The symptoms come on, not during the exposure to the increased pressure, but from half an hour to an hour after the return to the surface, and only if the individual has been previously exposed to the increased pressure for more than an hour. Those who are not used to such work seem especially liable to suffer, and there are also considerable individual variations in the degree of liability.

Pains in the limbs, and often in the joints, precede the onset of the paralysis, which is usually sudden. In a few minutes the legs may be powerless. Sensation is often lost as well as motion, and in all severe cases the sphincters are affected. The arms are seldom involved. If the paralysis is incomplete the power may return in the course of a few days, but in severe cases the palsy usually lasts for weeks or months, and may be permanent. Death may occur quickly from acute cerebral symptoms, or after some weeks from bedsores, cystitis, &c.

A careful examination of the spinal cord has been made in only two cases (Leyden, Schultze). In neither was there any trace of hæmor-

* Sorgenfrey, 'Neurologisches Centralblatt,' 1885, p. 198.

rhage, but in each there were indications of a slight disseminated myelitis in the dorsal region. These cases teach us nothing of the nature of the primary lesion. It is clearly related in some way to the diminution of the pressure to which the body has been exposed. It has been suggested that gases may escape from the blood and block up vessels, but it is difficult to see why this should occur especially in the spinal cord, and why such an effect should be delayed until an hour after the increased pressure is removed. Moxon has associated the effect with the imperfect blood supply to the lower part of the cord, and suggested that the long small arteries which pass along the nerves of the cauda equina may be compressed by the cerebro-spinal fluid. But it seems to be the dorsal and not the lumbar region in which the morbid process occurs. The pressure drives the blood from the surface, and probably lessens the amount in all vessels to which its influence extends. On the removal of the increased pressure the conditions must be reversed, perhaps in a degree proportioned to the duration of the pressure. Hence the paralysis is perhaps due to an acute revulsive anæmia of the cord, analogous to that which sometimes follows loss of blood. The dorsal cord, to judge by inflammation, is more prone to suffer acute disturbance of nutrition than any other part of the central nervous system.

The treatment most suitable to this disease is probably that for acute myelitis.

HÆMORRHAGE INTO THE SPINAL CORD : HÆMATOMYELIA.

Primary hæmorrhage into the spinal cord, sufficient to cause symptoms, is a very rare disease, and it is even more rare than is suggested by the cases now and then recorded as such. We have already seen that hæmorrhage may accompany myelitis; a considerable extravasation may occur when the inflammation is only commencing, during the stage of congestion, and while the symptoms are slight. Such cases are easily mistaken for primary hæmorrhage. The risk of error is not always removed by pathological observation. Inflammation results from hæmorrhage, and when indications of myelitis are found about a clot, it may be impossible to say whether these are primary or secondary. It is probable that many cases of secondary myelitic hæmorrhage have been regarded and described as primary, and it is possible that a few cases of primary hæmorrhage have been regarded as secondary. One writer, Hayem,* goes so far as to deny the occurrence of primary non-traumatic hæmorrhage; but such an exclusive

* 'Des Hémorrhagies Intra-rachidiennes,' Paris, 1872.

view is unwarranted. It is certain, however, that the history of primary hæmorrhage has been largely written from uncertain data, and will need extensive revision when a sufficient number of exact observations have accumulated.

ETIOLOGY.—The rarity of hæmorrhage into the cord is especially great in comparison with the frequency of hæmorrhage into the brain. The difference probably depends on the tortuous and long course of the arterial path to the cord, whereby the vessels are preserved from the high pressure which is the chief cause of the degeneration, dilatation, and rupture of the cerebral arteries. Miliary aneurisms are not found within the spinal cord. Hæmorrhage is far more common in males than in females. It may occur at any age, but is most frequent between twenty and forty. It has been seen in young children, even so early as seven months,* and many of the subjects have been in advanced life. It is possible that the apparent preponderance of cases between twenty and forty is due to the inclusion of many instances of myelitis, which is especially common at that period. Of immediate causes, injury is the most frequent, especially falls which involve a severe concussion of the spine; the spinal column may or may not be injured at the same time. Over-exertion and exposure to cold have in rare cases preceded the onset. Chronic alcoholism and sexual excess have been thought to predispose. In a case related to me by Dr. Radcliffe, an extensive hæmorrhage into the grey substance at the top of the lumbar enlargement, resulted from coitus four times repeated, the symptoms commencing suddenly during the fourth act. Minute extravasations are often found after death from diseases which interfere with respiration and cause venous congestion, and they are especially frequent in maladies which, at the same time, cause functional excitement of the cord. Thus they are common in tetanus and in convulsions. They cause no symptoms, and are probably produced during the last moments of life. They have been termed "*accessory*." The diseases of the cord that lead to *secondary* hæmorrhage are chiefly inflammation, tumours, and cavities in the cord. Thus we may distinguish four chief classes of hæmorrhage: primary, secondary, traumatic, and accessory.

PATHOLOGICAL ANATOMY.—The minute extravasations just mentioned, as met with after asphyxial and convulsive diseases, are found in both the grey and white substance, but especially in the former. They are usually microscopic, or visible to the naked eye as minute red points, distinguishable from distended vessels only by their slighter resistance to a stream of water. The extravasation may occupy the perivascular sheath, or the cavity in which the vessel lies, or extend between the nerve-elements. The larger non-traumatic hæmorrhages, which cause symptoms, always begin in the grey

* Clifford Allbutt, 'Lancet,' 1870, vol. ii, p. 84.

substance, and are often confined to it, extending into the white columns only when large in size. The vessels of the grey substance are more numerous than those of the white, have less external support, and probably undergo more considerable changes in state. The effusion forms a cavity in the cord, sometimes more or less rounded in transverse section, and half an inch or more in vertical extent. Sometimes the extravasation is irregular in shape. The cord is enlarged at the seat of the hæmorrhage, and this may be visible externally as a dark swelling the size of a nut or a bean. Very rarely the hæmorrhage tears the layer of cord which limits it, and blood, usually only in small quantity, escapes into the membranes. The tissue of the cord adjacent to the clot is usually broken down, stained, and softened, and inflammatory changes may be recognised in it with the microscope, just as in the neighbourhood of hæmorrhages into the brain. As in the latter, the effused blood slowly undergoes changes in tint, becoming first rusty and then yellow, and ultimately a cyst may remain. Several extravasations may coexist, usually in the same part of the cord. In contrast to this *focal hæmorrhage* there may be an infiltration of the grey matter with punctiform extravasations, which appear, until closely examined, to be a single hæmorrhage. The tissue between these small extravasations is broken down. It is probable that this form is always secondary to myelitis. In such secondary cases a careful microscopical examination usually reveals indications of inflammation much more extensive than the area affected by the hæmorrhage. A growth into which hæmorrhage occurs is usually a glioma, unaffected parts of which will be found in the neighbourhood of the extravasation. The cavities in the cord into which blood may escape are sometimes of considerable vertical extent. I have known a fissure in the posterior column to be filled with blood through almost the whole length of the cord.

SYMPTOMS.—Slight symptoms, “prodromata,” have been observed in some cases, chiefly in the form of trifling sensory disturbance, tingling, &c., in the limbs afterwards paralysed. They have existed for a few hours or days, or even for two or three weeks before the onset. It is probable, however, that these have been cases of secondary myelitic hæmorrhage, and that there are no premonitory symptoms in primary extravasations. The actual onset is always sudden; the symptoms attain a considerable height in the course of a few minutes. Occasionally there has been transient loss of consciousness without any cerebral lesion, or rarely the onset has been by a series of successive sudden augmentations of the symptoms. Sometimes the symptoms come on during sleep. The suddenness of the onset is the characteristic of the disease; the symptoms which thus develop vary according to the seat and extent of the extravasation. In the majority of cases there is paraplegia, complete motor and

sensory paralysis up to the level of the lesion, with loss of power over the sphincters. Pain commonly but not invariably accompanies the sudden palsy; it may be felt in the spine, or in the sacrum, or round the trunk, or in the legs. If in the spine it is local, and does not extend through a considerable length of the spine, as in meningeal hæmorrhage, and there is not the initial spasm and rigidity which characterise the latter disease. The spinal column may be tender at the seat of the hæmorrhage. Usually the paralysed muscles are relaxed. Sometimes they are the seat of early clonic contractions, or these may come on a few days after the onset. When the hæmorrhage is in the cervical region all the limbs are powerless; one arm is often affected before the other. The state of reflex action varies according to the seat of the disease; if it is at first abolished it quickly returns in the leg (unless the hæmorrhage is in the lumbar enlargement), and it soon becomes excessive. Vaso-motor and trophic changes in the skin are common; there is often vascular dilatation; bedsores readily form, and cystitis often results. Frequently the secretion of sweat is increased for a time. The temperature is normal at the onset, but it usually rises in the course of a few days from secondary inflammation in the cord.

The palsy developed at the onset usually continues for a week or ten days, although the pain may lessen. The symptoms do not always increase during the stage of inflammation, perhaps because this only involves the structures which are already impaired by pressure, but if the patient has been brought near to death by the primary hæmorrhage, the secondary inflammation may end life. Occasionally, moreover, symptoms of an ascending or descending myelitis may come on, and the former may cause death by its interference with the muscles of respiration. It is probable that this extension only occurs in cases of myelitic hæmorrhage, in which the extravasation is merely an incident in the course of a commencing inflammation. In cases of primary hæmorrhage the symptoms pass into a chronic stage, improvement being usually slow. Some lasting loss of power remains in most cases, and there is often some permanent muscular wasting on account of the frequency with which the hæmorrhage is in the cervical or lumbar grey matter. Occasionally there is rapid recovery up to a certain point; a hæmorrhage of small size may abolish conduction in the white columns by the suddenness with which it compresses them, and the effects of the pressure may quickly pass away.

DIAGNOSIS.—The diagnosis rests on the actually sudden onset of the symptoms. It must be remembered, however, that we cannot assume that symptoms which come on during the night's sleep are of sudden onset. Neglect of this consideration sometimes causes a mistake in diagnosis. The mode of onset is a sufficient distinction from all other organic diseases, except hæmorrhagic myelitis and meningeal hæmorrhage. The former is distinguished by the existence of slight

symptoms before the sudden attack. We are not justified in regarding as primary hæmorrhage any case in which premonitory symptoms existed for more than a few minutes, unless such symptoms were so pronounced and sudden in onset that they might have been due to a small extravasation. Initial fever in any case makes myelitis probable. The distinction from meningeal hæmorrhage has been mentioned in the account of the symptoms.

PROGNOSIS.—In all cases in which the symptoms are considerable in degree or wide in range, the danger to life is great, and remains great until they begin to subside. The prognosis is better when the disease is in the dorsal region than when it is in the enlargements. It is better when sensation returns in the course of a few days, but if the enlargements are affected other sources of danger remain considerable. Early trophic changes also render the prognosis worse. After the onset is over it is guided by the general principles that determine the prognosis in myelitis. ✓

TREATMENT.—The treatment of hæmorrhage into the substance of the cord is the same as that of hæmorrhage into the membranes (p. 209). Absolute rest is essential, and the prone position desirable. Ice should be applied to the spine opposite to the seat of the hæmorrhage. Full doses of ergot or ergotin may be given: 3ss of the liquid extract or five grains of ergotin may be given by the mouth, or three grains of ergotin may be injected under the skin, and the dose may be repeated two or three times, at intervals of two hours. A few large doses are probably more effectual than smaller doses continued for a longer time, since the hæmorrhage probably does not go on for long. The after-treatment must be that for myelitis.

DEGENERATIONS OF THE SPINAL CORD.

A large and important class of diseases of the spinal cord consists of those in which there is a slow degeneration of the nerve-elements, with an overgrowth of connective tissue, and in which structures are affected that have a common function, while others that have a different function escape even when they are adjacent to the elements that are diseased. Affecting thus functional "systems," they are termed "system diseases." This fact affords strong reason for believing that the primary change is in the nerve-elements themselves, and that the overgrowth of interstitial tissue is secondary. The process is thus analogous to that which occurs in the secondary degenerations, in which the first change is certainly in the nerve-elements, the destruction of

which is followed by an overgrowth of connective tissue, a "sclerosis" as it is termed. We have seen (p. 237) that the process is by some regarded as a slow, parenchymatous inflammation, an inflammation beginning in the proper functional elements of the organ. To term a process "inflammation," which proceeds so slowly that five or six years may pass before it attains a considerable degree (as is sometimes the case) and in which there is commonly no primary vascular disturbance, involves a change in our conception of inflammation; but so far as the process is concerned the question is one of name rather than of nature.

Another question of much greater interest is the relation of the two elements in the process, the wasting and the growth, the atrophy of the nerve-tissue, the hypertrophy of the connective tissue. The failure of nutrition in the one causes an increased energy of nutrition in the other. We have seen this relation in the nerve-fibres. The degeneration of a fibre is attended by an active growth of its nuclei and protoplasm. The nutrition of the two elements, the neural and the adventitial, is connected in the closest manner, but in inverse degree. It is important to recognise the fact that the process of growth of the connective tissue-elements is an active manifestation of nutritional energy, which, when once excited, may be to some extent independent of its cause. It may, when very active, pass beyond its original limits. It may, when very rapid, have some of the characters of an interstitial inflammation.

The degenerative diseases are not numerous, but they are of great importance. They are degeneration of the anterior cornua and anterior root-fibres, causing muscular atrophy; degeneration of the posterior columns and posterior root-fibres, causing locomotor ataxy; and degeneration of the pyramidal tracts, causing spastic paraplegia, a disease the exact pathological position of which is not quite certain. These degenerations may be variously combined.

In every form of spinal degeneration there is a tendency for the morbid process to extend beyond the system in which it begins and to which it is, for a time, confined. This tendency is much more marked in some degenerations than in others. It is apparently due to two influences. One is the tendency of the secondary interstitial process to independent extension, by which it may invade structures adjacent to, but distinct from, those which first undergo degeneration. But the extension cannot always be explained by contiguity. More distinct systems of structure may also suffer, apparently because the tendency to decay involves them also, although in slighter degree and at a later date than the elements first affected. In most degenerative diseases, in cases of severe degree and very long duration, almost all the elements of the cord may be in some degree involved.

LOCOMOTOR ATAXY (TABES DORSALIS: POSTERIOR SCLEROSIS).

The malady thus named is the most common chronic disease of the spinal cord. It consists in a degeneration in the posterior columns of the spinal cord, or the peripheral sensory nerves, or both, and is manifested, when considerable, by inco-ordination of movement, peculiar pains, and defective sensibility. The name "locomotor ataxy" was given to the disease by Duchenne; "tabes dorsalis," or "wasting of the back," is a term applied by Hippocrates to certain symptoms supposed to be due to venereal excess, and long ago limited in Germany to symptoms believed to depend on atrophy of the spinal cord. It then included all chronic paraplegias, but was further restricted to this disease by Romberg. It has lately obtained wider use on account of the discovery that inco-ordination may be absent when the disease is slight in degree.

The symptoms vary much in different cases. Besides the varieties thus produced, there are two forms which present many and important differences from that commonly met with. One is the so-called "hereditary ataxy." The other is that in which both weakness and inco-ordination co-exist from the first; "ataxic paraplegia" it may be called. Each differs from other varieties with sufficient constancy to merit separate consideration. These varieties are not included in the following account.

HISTORY.—The inco-ordination of movement and other symptoms of the disease were frequently noted, during the first third of this century, in cases of disease of the spinal cord, but such cases were not distinguished from those with actual loss of power. The inco-ordination was found to be associated with disease of the posterior columns by Stanley.* The first really exact account of the disease was published in 1847 by Todd,† who distinguished the cases with inco-ordination and without weakness, from simple paraplegia, and, apparently not aware of Stanley's observation, he inferred (from the character of the symptoms and his theory that the posterior columns contain fibres connecting segments of the cord at different levels) that the posterior columns would be found diseased; and he verified this inference by finding in two cases disease of these columns. The credit of the discovery of the disease belongs, if to anyone, unquestionably to Todd. Four years later (in 1851) Romberg described the disease and the lesion in the posterior columns, but he failed to exclude loss of power from the symptoms. Russell Reynolds in 1855 gave an accurate

* 'Med. Gazette,' Feb., 1840, and 'Med.-Chir. Trans.,' vol. xxiii.

† 'Cyclopædia of Anatomy and Physiology,' vol. iii, p. 721.

description of the symptoms, and was the first to attribute them to muscular anæsthesia.* A series of cases was described by Gull in 1856 and 1857. Türck first observed, with the microscope, the wasting of the fibres in the posterior columns. Duchenne in 1858-9 published an independent and very able analysis of the symptoms of the disease, and gave it the name "locomotor ataxy." He obtained for it (with the help of Trousseau) the recognition that previous descriptions had failed to secure.

CAUSES.—The disease is much more frequent, in this country at least, in urban than rural populations. Inherited influence is to be traced only in a small proportion of the cases, perhaps in not more than 10 per cent. It is usually a general neurotic heredity, manifested by the occurrence in relatives of such diseases as insanity, epilepsy, and other degenerative diseases of the nervous system. Instances of this are—father epileptic; father insane; two sisters insane. Direct inheritance of the disease is extremely rare (the special hereditary form being always excluded). A boy with distinct symptoms, and optic nerve atrophy, was the son of a man who presented characteristic indications of the early stage of tabes (Remak).

Males suffer far more frequently than females, the proportion being about ten to one. When every allowance has been made for the different incidence of causal influences, the fact seems to indicate a proclivity inherent in the male sex. The same preponderance of males obtains in a disease that has some alliances with tabes—general paralysis of the insane. The middle period of adult life is that in which the disease usually commences. No less than half the cases begin between thirty and forty, one quarter between forty and fifty, and rather less than a quarter between twenty and thirty. It rarely begins after fifty, but I have once known it to develop at sixty-six. Under twenty it is still more rare, but cases have been seen as early as ten and twelve.

Among the individual causes, one overshadows all the rest—the influence of syphilis. A very large proportion of the sufferers have had, at some previous time, constitutional syphilis, either distinct secondary symptoms or an indurated sore. The proportion is almost as large in the upper and middle classes as in the lower. Of fifty consecutive cases of the disease in men, seen in private consulting practice, no less than twenty-nine, or 58 per cent., gave a history of a chancre known to be hard, or of secondary symptoms; and eight others had had a venereal sore of unknown nature. In the lower classes the proportion is higher, and has been estimated at 70 or 80 per cent., or even more. When a deduction is made for possible accidental coincidence, there remains at least one-half of the cases in which numerical coincidence

* Reynolds, 'Diagnosis of Diseases of the Brain, &c.,' 1855, "Anæsthesia Muscularis." Certain words are worth quoting—"It appears most probable that the centripetal tract of fibres is affected; and that the locality of lesion is very variable."—P. 165.

must depend on causal relationship. It is probable indeed that, taking all cases, a causal proportion of two thirds would be nearer the truth. In women also antecedent syphilis can often be traced, although rather less frequently than in men. At the same time the full facts are difficult to ascertain, since in married women syphilis often runs a latent course. But in the cases of tabes that succeed syphilis the lesion is not syphilitic in histological character. In these, as in the cases in which syphilis can be excluded, the disease is a degenerative one. It is, moreover, rarely influenced by anti-syphilitic treatment. Hence it must be regarded as a degenerative sequel of syphilis rather than as a true syphilitic disease. It is probably the influence of syphilis that determines the greater incidence on the urban than on the rural population, and the preponderance of cases in middle life. Inherited syphilis also seems capable of causing the disease. In several cases of tabes in young persons, indications of inherited syphilis have been present. I have seen one example of this. The interval between the primary syphilis and the first symptoms of the disease varies from two to twenty years. It is, however, rarely less than five years, and in most cases it is between six and twelve years. Occasionally the malady develops as a sequel to some more distinctly syphilitic lesion of the cord.*

It is certain, however, that syphilis is not the only cause of the disease. In a few cases, perhaps 10 per cent. of the whole, it can be excluded with confidence. The causes operative in these can be traced also in some of the patients who present a history of syphilis, and in such cases the causation of the malady is probably complex.

One of these causes, which can sometimes be clearly traced, is injury, such as involves concussion of the spine. The immediate results of the injury, whether slight or grave, transient or lasting, are followed by the symptoms of the degenerative malady. In one case a man fell from a height on to the deck of a ship; transient paraplegia resulted, but six weeks after the accident the man presented extreme ataxy, with good power, and no knee-jerk (Arnold). I have known the symptoms to develop gradually a few months after a fall from a horse. Expo-

* Although an occasional relation to syphilis had been noted by several preceding observers, Fournier was the first (in 1876) to assert the wide extent of this relation. His statements were received with doubt, because syphilitic patients constituted his field for observation, but they were confirmed from the neurological side by myself, ('British Med. Journal,' March 1, 1879) and Erb ('Arch. f. klin. Med.,' July, 1879); abundant corroboration has been since afforded; many who at first doubted have been convinced by fresh observations. The difference in the character of the lesion from that of changes known to be syphilitic has been urged as an objection by many writers, but such theoretical considerations must yield to facts, and instead of denying that this or that lesion can be produced by a given cause, we may have to widen our view of the operation of that cause. Other degenerations of the nervous system seem to have a relation to syphilis, and, as I wrote some years ago, "it seems that one effect of constitutional syphilis may be to induce a neuropathic state in which certain degenerative diseases of the nervous system readily occur" ('Lancet,' Jan. 15, 1881).

sure to cold and wet has occasionally preceded the onset so directly that it must be regarded as a cause. In other cases the disease has succeeded excessive fatigue and over-exertion, and also certain acute diseases, especially acute rheumatism and typhoid fever. The latter is known to cause focal myelitis; except by such an agency acute diseases can hardly do more than excite to activity a pre-existing tendency. Alcoholic excess has been noted in rare cases, and the influence of this cause is intelligible since it is known to cause neuritis, and sometimes such a form of neuritis as occurs in tabes. Indeed it is strange that this cause cannot be traced more frequently. Sexual excess has been supposed, by some, to be a cause; but its influence can rarely be traced, and its significance is uncertain, since sexual excitement is undoubtedly sometimes an early symptom of the disease.

Secondary Tabes.—Symptoms of locomotor ataxy sometimes succeed other diseases of the spinal cord, and such sequence is especially common in syphilitic subjects. Myelitis and syphilitic gummata may be thus succeeded by tabes. It is not surprising that a degenerative tendency, effective alone, should be determined in its operation by local disease. Thus an officer in India, who had had syphilis, having suffered for a day or two from pains in the back, took a bath in snow water, and in a few days his legs were absolutely powerless. He gradually recovered power, but could not co-ordinate the movements: as power returned, lightning pains came on, and a year afterwards he presented the typical condition of locomotor ataxy. Again, a man, twelve years after syphilis, had a severe fall, followed by gradual loss of power, so that at the end of three weeks he could scarcely stand. The legs remained weak for a month, and then improved, but ataxy came on; three months later, power was good, inco-ordination extreme. A similar succession may occasionally be observed in those who have not had syphilis. In many cases, however, muscular strength does not rise to the normal standard, and the condition is one of combined weakness and ataxy.

In recognising the fact that tabes is occasionally secondary to a primary myelitis, it is necessary to note that myelitis may also occur in the course of tabes. We must, therefore, ascertain the absence of preceding tabetic symptoms before the disease can be regarded as purely secondary.

SYMPTOMS.—A typical case of developed tabes presents certain motor, sensory, and reflex symptoms. There is inco-ordination of movement of the legs, sometimes of the arms also, without loss of power or muscular wasting. There are pains in the affected parts, especially sharp momentary “lightning” pains; there is some loss of sensation; there is often loss or diminution of reflex action from the skin, and almost always entire loss of the myotatic irritability that is revealed by the so-called “tendon-reflexes,” and especially by the knee-jerk; there are retention or incontinence of urine, constipation, and often

loss of sexual power. Of this group of symptoms, two usually precede the others—the pains, and the loss of the knee-jerk. These may exist alone, even for years, before inco-ordination comes on. Thus the symptoms are far wider in range than the name “ataxy” suggests, and while inco-ordination, if it exists, is the most obtrusive objective symptom, it is not the essential symptom of the disease, and, indeed, may never be developed. Hence physicians have extensively fallen back on the older term “tabes dorsalis.” But recent expansion of our knowledge, especially the discovery of the extent to which the symptoms may depend on disease of the peripheral nerves, shows that even the qualifying “dorsalis” narrows the term within the pathological conception of the disease.

Besides the symptoms above enumerated, others are occasionally present. Of these the most important are atrophy of the optic nerve, and occasionally of other cranial nerves, trophic changes in the skin, the bones and the joints, and an ultimate extension to motor paralysis and muscular wasting. The combinations of symptoms present in different cases vary much. It will be convenient to consider first the symptoms in their various degrees, and then their grouping and sequence.

Motor Symptoms.—The characteristic inco-ordination of movement develops gradually. It is always increased (as Romberg first pointed out) by closure of the eyes, and at first may only exist when the guiding influence of vision is thus withdrawn. Before it causes ataxy of movement, it may render difficult the maintenance of equilibrium when the base of support is narrowed by the feet being placed close together, toes and heels. If then the eyes are closed, the patient sways, and may even tend to fall. In health slight unsteadiness is thus produced, varying in degree in different persons, but never amounting to even a suggestion of a fall. The effect of closure of the eyes is greatest when sensation in the soles of the feet is defective, but does not depend on this loss; it may be marked when sensation on the soles of the feet is perfect. The early defect in co-ordination may be discovered by the patient when he walks in the dark, or, not uncommonly, when he shuts his eyes in the process of washing the face. In a further degree of inco-ordination there is inability to stand with the feet together even when the eyes are open, and the patient is only steady when the feet are wide apart. If the feet are bare, the difficulty is greater, because muscular action has to replace the rigid base of the boot. The irregular contraction of the muscles is shown by the conspicuous movement of the tendons on the back of the feet. The patient may oscillate from toes to heels before he comes to rest. As the defect progresses, uncertainty is felt in walking even with full visual guidance, especially on uneven ground, or on a very smooth surface. A slight visible alteration in gait is then appreciable, the feet are not placed on the ground quite as in health, or there is distinct difficulty in maintaining equilibrium when the patient turns quickly. He has to

put a foot down suddenly to keep from falling. The alteration is greater when the eyes are closed, and is especially conspicuous on an attempt to walk backwards—a test that I have seen employed by Sir William Jenner many years ago. As the inco-ordination increases, the change in gait becomes greater, but varies much in its precise characters, probably according to the muscles that are most affected. Often the feet are raised too high, thrown forwards too far, brought down too suddenly, and the whole sole comes in contact with the ground at once. There may be a tendency for one foot to be brought in front of the other, or, in avoiding this, the feet may be moved too far outwards. Often the foot becomes inverted when it touches the ground. Efforts to correct error in movement have themselves to be corrected. In other cases the defect in maintaining equilibrium is greater than the disorder of movement; the patient sways about in the manner of one who has cerebellar disease. As the defect progresses, the patient is only able to walk by steadying himself with a stick, or by taking hold of the arm of another person, or of adjacent objects. At first a very slight degree of this help is sufficient; guidance rather than support is needed. Afterwards, however, considerable support is necessary, and ultimately the patient may be unable to stand even with help. When he attempts to rise, the legs move hurriedly forwards and backwards, and if the upright posture is at last achieved the legs slip forward, and only strong support saves the patient from a fall. The ataxy is manifest also in other movements of the legs. If the patient, when lying, tries to touch an object with his foot, the leg is moved irregularly, goes beyond the place, and then is brought too far back, and only at last does it come in contact with the object, often with unintended force. This inco-ordination, like that in standing, is much greater if the eyes are closed.

The arms may present similar inco-ordination, although they may escape, even when the affection of the legs is extreme. Sometimes both legs and arm are deranged together; very rarely the arms suffer before the legs. The commencing defect is first revealed by delicate movements, such as writing. When slight it may be conspicuous if the patient tries, with closed eyes, to touch some object, such as his own nose, or, having abducted his arms, tries to bring the forefingers together. As it increases, all movements become irregular; it is impossible for the patient to button his coat, or to pick up a small object from the table; the fingers twist about in the attempt, and the grasp is not sustained; first one finger is felt to relax and then another. If the patient attempts to hold out his hand in a fixed posture it is seen that the same irregularity obtains; instead of a uniform balanced contraction the muscles contract and relax involuntarily, and slow unintended movements of the fingers result (Fig. 106), sometimes closely resembling those of athetosis. The same spontaneous movements may also be observed in the legs. They cease at once when the muscular effort is relinquished. Occasionally the muscles of the

trunk present a similar ataxy. Thus one patient could sit steadily on a chair when his eyes were open, but if he closed them would at once fall off. In such a case, if the legs are but little affected, there may be (as I have seen) scarcely any inco-ordination of movement of the legs, but extreme unsteadiness and reeling on an attempt to stand or walk. The movements of the head, face, tongue, and eyes, always escape derangement.

Even with extreme inco-ordination, motor power may be unimpaired. Occasionally some group of muscles, as the flexors of the ankle, become weak for a little time and then strong again, just as may the eyeball muscles, as we shall presently see. In some cases motor power in the limbs remains unimpaired to the end; more often, when the ataxy has become great, some muscular weakness supervenes, with or without wasting of the weak muscles. There is a distinct group of cases in which weakness and ataxy come on together, but these are considered separately ("Ataxic Paraplegia").

Sensory symptoms are prominent in most cases. They consist of subjective sensations, chiefly of pain and loss of sensibility. Spontaneous pains are present in some degree in nine-tenths of the cases of tabes. The most frequent and characteristic are the sudden and lancinating pains called "lightning pains." They occur chiefly in the legs, but may be felt in the trunk, arms, and even in the head. They are usually paroxysmal; attacks of such pains last for some hours or for a day or two, varying in seat, but often felt in the same part throughout an attack. Sometimes they are felt in a limited area of the skin, sometimes they seem more deeply seated, sometimes they dart down the limb. They may correspond to a nerve-trunk, but more frequently have no relation to the nerves. Although the pains are usually acute and "stabbing," they are sometimes of different character. Thus in one patient the sensation was as if both legs were, for a moment, on fire. When referred to the skin, this often becomes tender, so that even the contact of the bedclothes cannot be borne. When pains continue at one spot for some days, ecchymoses have been known to form there. In one case, after pains had been felt at one part of the scalp for a time, the growth of the hair was changed; each hair bent and broke short off, over an area the size of half-a-crown; after the pains ceased the growth of the hair became normal. A sharp pain may be accompanied by a sudden reflex spasmodic movement of the legs, or by inhibitory weakness. Thus, in one case, a sudden pain would often make the patient fall on his knees. The pains may be so severe as to prevent sleep for several days.

Other kinds of severe pain, less brief than the lightning pains, occur in some cases, and are described by various epithets, as "burning," "tearing," "gnawing." Burning pain in the toes distressed one patient. Much more common, and occurring with other pains, are slighter dull pains like those of rheumatism, for which they are often mistaken, an error that is facilitated by the circumstance that any of

the pains of tabes may be influenced by weather, being especially increased by damp cold. A painful sense of constriction is felt by some sufferers in the legs, groins, genitals, or trunk; it is less common than in myelitis, but when it does occur it is sometimes very intense; the patient may feel as if the whole trunk were tightly enveloped in a cuirass of brass. In the hands pains are common, but are usually slight in degree, and are occasionally felt chiefly in the ulnar area. Visceral pains may also occur, usually paroxysmal, and referred to the stomach, bladder, rectum, &c. These will be again considered in connection with the visceral symptoms of tabes. Pains are not only the most common, they are often the earliest symptom of the disease. Nevertheless they are sometimes slight, and occasionally are altogether absent. Perhaps they sometimes occur alone; I have seen two patients who suffered characteristic lightning pains, but presented *no* other symptoms of tabes; the tabetic nature of the pains is therefore uncertain.

Sensations other than pain are also common. They are various in character, described as "tingling," "pins and needles," "creeping," and very often as if the skin, especially of the soles, was covered by some soft substance. Sensations of cold or of heat are also common; the feet may feel as if the legs were always in cold water; a sensation of cold about the testicles is occasionally complained of. The sense of heat may be so intense as to amount to pain, as already mentioned. Similar cutaneous sensations are also common in the hands. Increased sensitiveness of the skin, chiefly to pain, may also exist, especially on the soles of the feet, and often accompanies lessened sensibility to touch.

A diminution of sensibility is also present in most cases of the developed disease. It may involve all forms of sensibility, or one much more than the others, or some forms may be much impaired and others normal. It is equally common for either pain or touch to be affected alone; for instance, a prick may cause no sensation of pain, although the least touch of the finger is everywhere perceived. A firm touch (*i. e.* slight pressure) may be felt when a slight touch is unperceived. When a touch is felt, the power of localising it may be impaired. Temperature sensations are rarely affected without other forms of sensibility, but they may be impaired with pain when tactile sensibility is normal. On the other hand, there may be extensive loss to pain without any defect of the temperature sense. A prick may then cause only a sensation of heat, and extreme degrees of heat or cold cause pain less readily than normal. The temperature loss may be partial—either cold or heat may be unperceived while the other is recognised. When touch is slightly impaired there may be an inability to discriminate differences in the degree of pressure on the skin.

Accompanying the diminution or increase of sensitiveness there are sometimes curious changes in the sensation produced. One of these is a delay in the perception of pain, which may amount to several

seconds. The greatest delay I have met with amounted to seven seconds, but one of fifteen seconds has been described (Eulenberg). The contact of a point may be felt at once, the pain only after a considerable delay. Often there is an after-pain lasting sometimes for a quarter or half a minute or longer, and the maximum sensation may not be attained until some seconds after the pain is first felt. Thus, in a case in which there was a delay of seven seconds, the maximum sensation was only felt twenty-five seconds after the prick (Obersteiner). A second increase of pain has been noted (Naunyn). I have known a sort of rhythmical recurrence of sensation; the pain was not felt for several seconds, and then the sensation was repeated several times in successive darts of pain. A delay greater than normal may also attend the sensation of heat. The localising power is sometimes strangely perverted. A touch or prick on one leg may be referred to the other, sometimes with singular uniformity in position (allocheiria). A prick in one spot may be felt in many places (polyæsthesia) on the same, or (as I have seen) on both legs. It is said that the reaction of the sensory nerves to electricity may be changed in the same way as that of the motor nerves is changed when they are degenerated. Instead of the earliest sensation occurring on closure of the circuit, at the negative pole (kathode), as in health, it may be felt first at the positive pole (anode). (Mendelssohn.)

The region in which sensibility is impaired varies much in different cases. The loss occurs most frequently, and is usually greatest, in the lower part of the legs and feet. It may be limited to the soles, and usually then ceases at the outer sharp edge of the foot, but extends for an inch or so up the inner side. Touch or pain may be lost on the sole only, and even, strange to say, the form of sensibility that is lost on the sole may alone persist in the legs. Although the sole is usually most affected, sensation may be perfect on the sole when lost elsewhere. One leg is often more affected than the other. The loss may extend to the trunk or even the neck. In the arms it is usually greatest on the hands, and often commences on the palmar aspect of the fingers. Sensation may be lost on the trunk and not on the limbs, an important fact, because the anæsthesia may then readily be overlooked. On the head a loss is rare, but has been noted on the cheeks.

The loss of sensibility is not confined to the skin, but affects the deeper tissues also in many cases. The contraction of the muscles on electrical stimulation may be unfelt, and the muscles may be insensitive to pressure or forcible extension. It is probable that the sensibility of the joints, fibrous tissues, and tendons is also involved. The position of the limbs in passive as well as in active movements may not be perceived. In slight cases this sense of posture may not be lost, but in severe cases it may be so absolutely abolished that one of my patients, who often fell out of bed, had always to look to see where his legs had gone to before he attempted to rise. The anæsthesia may extend to the viscera when the trunk is involved. In conse-

quence of the loss of sensation grave injuries to the limbs, such as burns, may be unperceived, and visceral disease may be unattended with the customary pain. I have known a severe attack of pleurisy to be absolutely painless.

Cutaneous anæsthesia, although it is thus a common, and often an early symptom, is not invariable. Every test may fail to reveal any loss, and this integrity of sensation may exist in patients who present considerable inco-ordination, and in those who suffer severe pains, as well as in those who have had no pains. But it is very rare to have no sensory loss when inco-ordination is considerable.

Reflex action from the skin is usually impaired in proportion to the loss of cutaneous sensibility, and especially to the loss of tactile sensibility. It may be normal when pain is unfelt. But reflex action may be lessened when there is no loss of sensation. The plantar-reflex is most frequently impaired, and the progress upwards of the disease may be attended by a progressive loss of the gluteal, cremasteric, and abdominal reflexes. On the other hand, in the early stage of the disease, there is sometimes a very marked excess of all cutaneous reflex action, and this may reach a degree rarely met with in any other affection. In one case a touch on the sole would make the foot fly up, and if the naked trunk was stroked, so violent a start was caused that the patient on one occasion rolled off a sofa. In this patient considerable ataxy coexisted with the excess, but the reflex over-action is usually an early symptom, and passes away as the ataxy becomes well developed.

The sexual act is, in part, a superficial reflex action, and increased sexual excitement sometimes coexists with the excess of reflex action. In the case just mentioned there was intense satyriasis. In the same manner the sexual power usually fails when the superficial reflex actions subserved by the corresponding region of the cord are abolished—the cremaster and the lower abdominal reflexes, but to this rule exceptions occur. Loss of sexual power is an exceedingly common and often an early symptom of the disease. It may precede any impairment of sensory or reflex functions. On the other hand, exceptions are met with, in which sexual power persists to a late period of the disease, even until after the patient has lost the ability to walk alone.

If the condition of cutaneous reflex action is somewhat inconstant, this is not the case with the reflex process on which the myotatic irritability depends. A loss of the knee-jerk, as Westphal first pointed out, is one of the earliest and most constant symptoms of tabes. With it disappears also all other indications of this irritability. In cases with great excess of cutaneous reflex action, the knee-jerk is lost as in other cases, and the contrast between the two forms of reflex action is very striking. It is exceedingly rare for the knee-jerk to be obtained in any case of true tabes. Very rarely an early case may be met with in which it is not lost, although diminished, and commonly unequal on the two sides. In one or two cases I have found it slight

on one side and lost on the other when distinct ataxy was present, and have observed its speedy entire disappearance. In one case, in which the jerk was lost in one leg only, lightning pains were confined to this leg. The loss often precedes for many years the development of inco-ordination. But the muscles preserve their irritability to percussion, the contraction thus produced being limited to the part struck. In cases of ataxic paraplegia, which are often confounded with true tabes, the knee-jerk is not lost, and is usually excessive, and these, for the most part, constitute the cases sometimes described of locomotor ataxy without loss of this phenomenon.

Eye Symptoms.—The functions of the internal muscles of the eye are often affected in tabes. In no less than four-fifths of the cases the reflex action of the iris to light is lost. In most of these the contraction on accommodation is preserved—a characteristic first noted by Argyll-Robertson. Often also (as Erb has shown) the dilatation that occurs on painful stimulation of the skin of the neck, &c., can no longer be thus produced. Sometimes the ciliary muscle is also paralysed, causing loss of accommodation. Very rarely there is loss of accommodation and of the associated contraction of the iris, without loss of the light-reflex. The pupils are often very small (“spinal myosis”), especially when there is loss of the reflex dilatation from the skin, a loss which seems to be accompanied by atony of the radiating fibres supplied by the sympathetic. The pupils are not always small; they may be of medium size or beyond medium size, and then I have usually found that the skin-reflex can be obtained, although the light-reflex cannot. They are often not perfectly circular, and are sometimes unequal. Both eyes are usually affected in the same manner; occasionally the reflex action is lost in one and only lessened in the other. On the other hand, the action of the iris is sometimes perfectly natural, even in advanced cases.

Paralysis of the external ocular muscles is also common in tabes, and occurs in several forms: (1) Transient weakness, lasting a few days or weeks and then passing away. (2) Permanent paralysis, complete or incomplete, of a single nerve, or part of a nerve. Either form may occur at any stage, but the first is most common in the



FIG. 106.—Double tabetic ptosis.*

* From a Salpêtrière photograph, for which I am indebted to M. Charcot.

early, and the second in the later stages of the disease. The transient palsy renders diplopia a common symptom in the early period; any muscle may be affected but the external rectus is that which most frequently suffers. The persistent palsy may affect one or more muscles; often the levator is involved, causing what has been termed "tabetic ptosis" (Fig. 106). Sometimes the whole third nerve is paralysed. (3) There may be a combined palsy suggesting a cerebral origin. Rarely there is loss of the movement of convergence associated with loss of accommodation, although the internal recti may act in the movement towards one side. (4) Many or all the muscles of both eyes may gradually become paralysed, "external ophthalmoplegia," in consequence of disease of their centres.* This is very rarely met with in tabes, although it seems to bear a similar relation to syphilis.

Atrophy of the optic nerve occurs in tabes, and this disease furnishes a large proportion of the cases of simple atrophy. Its frequency is difficult to ascertain, but is certainly less than is often supposed. It does not occur in more than one case in ten. It is generally an early symptom, usually commencing before inco-ordination is developed, and in a large number of cases ataxy never comes on, the spinal malady becoming stationary when the nerve suffers. It rarely begins after the gait is considerably disordered.

The failure of sight usually commences with a peripheral limitation of the field and loss of colour vision, but sometimes central acuity fails early. When this remains good, the peripheral loss may reach a considerable degree before it is suspected by the patient. The visible pallor of the optic disc is not always proportioned to the failure of sight. The change in tint depends on wasting of the capillaries, and this is not always proportioned to the wasting of the nerve-fibres. Ultimately the disk is greyish in tint, and the retinal vessels are but little narrowed.† Occasionally there are slight signs of congestion in the early stage of the atrophy, and then some tissue of gelatinous aspect may develop in the disc, and the vessels may be narrowed.

The course of the atrophy is usually slowly progressive, and, in most cases, it ends in total or almost total blindness. It may, however, cease to progress, and even undergo slight improvement. The onset is not always gradual. Sometimes sight fails considerably in the course of a few days without ophthalmoscopic changes to account for it. I have met with one case in which temporal hemiopia coming on rapidly suggested disease at the optic chiasma. The atrophy is often more advanced in one eye than in the other, and very rarely one eye only suffers. Flashes of light are occasionally experienced in the course of the atrophy, comparable to the lightning pains in the limbs, but they are not common. Headache is occasionally troublesome in cases of tabetic atrophy, and cephalic lightning pains are sometimes severe. Organic cerebral disease may be thus suggested.

* Buzzard, 'Clin. Lect. on Diseases of the Nervous System,' 1882, p. 180.

† Further details will be found in 'Medical Ophthalmoscopy,' 2nd ed., p. 163.

Deafness, having the characters of nerve-deafness, is met with in some cases, sudden or gradual in onset, transient or lasting. The persistent deafness has been attributed to an atrophy of the auditory nerve, analogous to that of the optic nerve, but certainly in many cases without sufficient reason. It is very difficult to distinguish an affection of the nerve from one of the labyrinth independent of the nerve. Even in a case of bilateral deafness coming on in the course of tabes, in which the patient could hear only a loud voice, and deep notes better than high ones, in which atrophy had been diagnosed, Lucae found calcareous masses in each of the labyrinths, and the auditory nerves were quite normal. But if there is a progressive limitation of the range of hearing, analogous to the peripheral limitation of the field of vision, we are justified in assuming nerve atrophy. I have seen two cases of this kind. In each there was also optic nerve atrophy, and the other symptoms of tabes were distinct. In one only notes between E^2 and D could be heard.* In the other the patient, when first examined, was absolutely deaf to notes above E^2 and below G_1 . A few months later the restriction had confined the range of hearing to the octave in the treble clef, between E^2 and E^1 , even Eh^1 being inaudible. Thus the loss occurred chiefly from below upwards. Ultimately no note could be heard. Attacks of vertigo are frequent in the cases attended by deafness, and seem to depend on the disturbance of the labyrinth or auditory nerve, and to be thus connected only indirectly with the primary disease.

The functions of the other cranial nerves are not often affected. Pains may be felt in the region of the fifth nerve. Unilateral atrophy of the tongue has been noted in one case. In the larynx, besides the spasm to be presently described, paralysis of the vocal cords has been met with in rare cases, both as an isolated symptom and also after attacks of spasm have occurred for a long time. Both posterior crico-arytænoids have been paralysed (Oppenheim), and in one case paralysis of one posticus occurred very early in the disease (Remak). A sort of ataxic irregularity in the movement of the cords has been described by Uthoff.

Sphincters.—The functions of the bladder are frequently deranged; there may be either sluggish micturition or a tendency to incontinence, and often the bladder is not perfectly emptied. These symptoms frequently occur early in the disease, and it may be indeed only for these that the patient seeks medical advice. Occasionally there is a curious reflex susceptibility on the part of the bladder; thus the urine always escaped from one patient when his hands were placed in cold water. The retention may become absolute, or there may be overflow incontinence; there is rarely paralytic incontinence. More commonly the affection of the bladder remains slight in degree, although even

* The seven notes above the middle C of the bass clef are designated by simple letters, those of each higher and lower octave are indicated by small figures placed above and below the letter. Thus the middle C of the treble clef is C^2 .

this may have serious effects. If the bladder for a long time is imperfectly emptied, cystitis is apt to occur, and secondary renal disease is a not uncommon cause of death when the difficulty in micturition has been insignificant. The sphincter ani is often also weak, so that a loose stool can be retained with difficulty, but its paralysis rarely reaches a high degree. Constipation is extremely common.

Vaso-motor and trophic disturbances constitute a singular group of symptoms to which much attention has lately been drawn. Transient œdema of the limbs may occur without albuminuria, and is sometimes an early symptom. Local sweating has been noted, confined, for instance, to the palms and soles, or to one side of the head. The ecchymoses and altered growth of hair, in connection with attacks of pain, have been already mentioned. Pigment may disappear from the skin and hair in isolated spots. Herpes of the skin is not uncommon, and usually occurs in association with attacks of pain. I have twice known the penis to be the seat of herpetic eruptions. The epidermis of the sole becomes thickened; blisters readily form beneath it and may lead to indolent ulcers. The curious affection known as "perforating ulcer



FIG. 107.—Perforating ulcer of the foot in tabes. (From a Salpêtrière photograph.)

of the foot" has been found to be almost confined to cases of tabes. Troublesome ulceration of the toes, especially about the nails, is also occasionally met with, and may necessitate amputation. The growth of the nails of the feet, and sometimes of those of the hands, may be changed. They may be thickened and the surface furrowed or uneven. The nails may even fall off and be slowly renewed. The teeth sometimes decay quickly, and may drop out. I have seen one patient, who had previously lost many of his nails, in whom all the teeth of the upper jaw fell out in the course of three days, without any pain or decay of the teeth. Wasting of the muscles may occur in the later stage of the disease, usually greatest towards the extremity of the limbs, and sometimes so great as to resemble that of progressive muscular atrophy.

Changes in the nutrition of the joints and bones have attracted

much notice since attention was directed to them by the careful investigations of Charcot. They are not very common, but are sufficiently frequent and well marked to place their relation to the disease beyond doubt. The bones may become brittle, and may break readily in so-called "spontaneous" fractures. The process of union is attended with the formation of a large amount of callus. Sometimes there is ossification of the tissues adjacent to injured bones. In the joints the changes consist of erosion of the cartilage, wasting of the heads of the bones, ossification of the ligaments, and sometimes irregular bony growths. The movement of the joints may be thus too free or too limited. The wasting of the head of the femur renders dislocation of the hip easy. A remarkable instance of these osseous changes has been recorded by Charcot; a tabetic woman suffered successively, in the course of a few years, dislocation of both hips, fractures of the left femur, of both bones of each forearm, and dislocation of one shoulder; after death old fractures were found in both scapulæ and the left clavicle. With these changes, and often when they are slight in degree, there is effusion within the joints and occasionally œdema outside them. The larger joints are those most commonly diseased—knee, hip, ankle, elbow—but the small joints of the

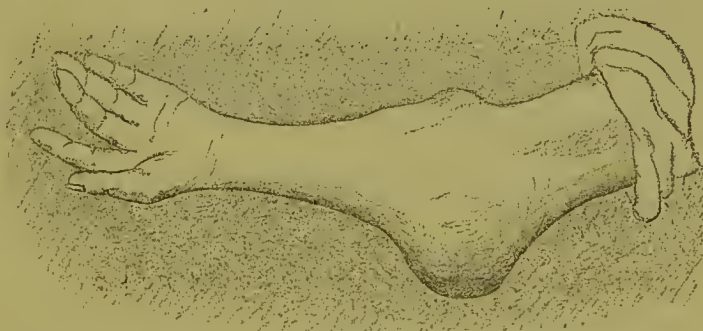


FIG. 108.—Locomotor ataxy, painless swelling of elbow-joint after a fall on it. One condyle of the humerus and olecranon were found, after death, to have been broken off. Several separate pieces of new bone had formed in the capsule of the distended joint.

fingers have been known to suffer (Westphal). The lesions are sometimes excited by injury, and the extraordinary changes that may follow traumatic influence is alone a conclusive indication that abnormal trophic influences are at work. Fig. 106 represents the arm of a man who fell and struck his elbow, fracturing the olecranon and condyle. It was followed by extreme painless swelling, and bony masses could be felt, gradually increasing in size. After death the fractures of the bones were found, and masses of new bone had formed in the capsule of the joint.

The origin of the changes in some joints, as the knee, is more complex. This joint depends for its functional capacity partly on its ligaments, and partly on the support of the muscular tendons that pass

by it, in one of which the patella is inserted. The sudden variations in the contraction of the muscles both render this support variable and determine sudden strains on the unsupported ligaments. Under this strain they yield, and when the patient stands the joint may become retroflexed as in Fig. 109. The strain on the ligaments and capsule leads to effusion, and may excite the more serious trophic changes



FIG. 109.—Locomotor ataxy; retroflexion of knee-joints.

already described. Changes in the tarsal bones and articulations may cause the foot to become flat, with a projection inwards or backwards of the tarso-metatarsal articulation, and of the tarsal bones. The condition has been called the "tabetic foot," by Charcot and Féré.*



FIG. 110.—Tabetic foot (from a photograph by Mr. Marriott, B.Sc.).

Visceral symptoms of peculiar character occur in many cases of tabes.† They consist almost entirely of paroxysmal disturbance of function, usually attended with great pain, and have been termed, by the French, *crises*, with qualifying adjectives according to the seat of the symptom. There is a tendency to over-elaborate this terminology. The most frequent seat of such disturbance is the stomach, and these attacks are called *gastric crises*. They consist of paroxysms of severe gastric pain, felt in the epigastrium and often passing through to the back. The pain is accompanied by vomiting, with or without nausea. The vomiting is often incessant, and is first of food, then of clear liquid, which may be very abundant; ultimately bile is vomited, and sometimes blood. Retardation or irregularity of

* The trophic changes in bone and joint have been regarded by some as simple chronic osteo-arthritis, without causal relation to the nerve disease in their subjects. All evidence seems to me opposed to this view. The arguments that can be adduced in support of it will be found in some of the speeches delivered at a discussion at the Clinical Society, Nov. and Dec., 1885.

† The occurrence of attacks of vomiting, sometimes early in the disease, was pointed out by Topinard, but thought by him to be a mere complication. (Topinard, 'De l'ataxie locomotrice,' Paris, 1864, p. 273.)

the action of the heart has sometimes attended such attack, and, rarely, pyrexia has been noted. I have known frequent hiccough to accompany the vomiting. Such an attack lasts for some hours or days, and then subsides, to recur in a few weeks. During the intervals the functions of the stomach may be performed in a perfectly normal manner. Although pain and vomiting usually occur together, some patients have attacks of pain without vomiting, and in others there is vomiting without pain. Rarely there is nausea alone. A temperate man, in the early stage of tabes, suffered during three years from frequent attacks in which, for several days, he had intense nausea each morning, passing away in the afternoon, and succeeded, in the evening, by an inordinate craving for food. He never vomited unless he made himself do so, in the vain hope of thus obtaining relief. After two years attacks of laryngeal spasm were added to the attacks of nausea.

The chief *intestinal* disturbance is constipation, but paroxysmal diarrhœa has been supposed to be of vaso-motor origin, and connected with the disease (Pierret). Paroxysms of rectal pain (*rectal crises*) are not uncommon, and are often accompanied by distressing tenesmus, sometimes by a sensation as if there was a foreign body in the rectum. In rare cases, paroxysms of pain have precisely the character of attacks of renal colic (*nephralgic crises*), or are felt in the neck of the bladder or along the urethra or at the meatus (*vesical or urethral crises*). These may be attended by an intense desire to micturate, although the bladder may be empty and only a few drops of urine may be expelled. In one patient such pain was repeated several times an hour, and each attack lasted for several days. Blood may be passed after an attack. Paroxysms of sexual excitement in women have been somewhat unnecessarily termed "*clitoris crises*."

"*Laryngeal crises*" are, perhaps, after those of the stomach, the most common of these symptoms. They vary much in character. The most common form is a true laryngeal spasm, with noisy inspiration and expiration, cough, and often considerable dyspnœa. The paroxysms may resemble those of whooping-cough or of laryngismus stridulus. Pressure on the superior laryngeal nerve at its entrance into the larynx, or on the trachea, or the introduction of a sound, will sometimes induce an attack. The spasm may last for a quarter of an hour or for some hours, but rarely continues so long as the gastric crises. In one recorded case the spasm spread to the pharynx, making swallowing impossible; a very violent attack extended to the muscles of respiration and the patient died asphyxiated. Death from these attacks is, however, extremely rare. Paroxysms of rough cough have been termed "*bronchial crises*;" in one case such attacks ceased when the patient began to suffer from gastric crises. These crises are often early symptoms, and they may continue for many years. I have seen a patient, still in the first stage, who gave a history of gastric crises during the preceding eighteen years.

Comparable to these paroxysmal visceral disturbances, although

very different in character and more alarming in aspect, are cerebral symptoms, which are, fortunately, very rare. They resemble the attacks that are common in general paralysis of the insane, and consist of transient apoplectiform seizures, hemiplegia, or convulsions general or one-sided. They may occur early in the course of the disease. The alarm they occasion is not without foundation, since death has been known to occur during an attack of apoplectic semblance. Paroxysms of vertigo sometimes seem to be of similar nature, but they more often depend on derangement of the auditory nerve.

Course and Termination.—It is convenient to divide the course of the disease into three stages :—(1) In which there is no alteration in gait, the chief indications of the malady being the loss of the knee-jerk and the pains, often associated with loss of the light reflex of the iris, and unsteadiness on standing with the feet together and eyes shut. (2) That in which there is distinct affection of gait, slight or considerable, but in which the patient is still able to walk, alone or with the aid of one or two sticks. (3) In which walking is possible only with the aid of another person, or it is impossible for the patient to walk or to stand. The first stage is not always present. Inco-ordination may be the earliest symptom, and I have known, in one or two cases, characteristic ataxy to be developed before the knee-jerk was lost.

The course of the symptoms is extremely variable. The epithet “progressive” given to the malady by Duchenne expresses a characteristic of a large proportion of the cases in which ataxy is developed, and he knew no other cases of the disease. The inco-ordination tends to increase, slowly or rapidly, until it reaches a considerable degree, and the sensory loss shows a like tendency. The pains fortunately do not always progress with the other symptoms. They are often most severe in the early period of the disease, and may either lessen or continue unchanged as the loss of sensation increases. But the power of recognising the first stage of the disease, which we owe, especially, to Westphal’s discovery of the loss of the knee-jerk, has enlarged, and in enlarging has to some extent modified, our conception of the general tendency of the disease. It is exceedingly common for the first stage to remain stationary for a long time, for ten, twenty, and even, in one case, for twenty-five years, if the duration of the lightning pains may be accepted as proof of its existence. When all cases are taken together and patients are subjected to careful treatment, I do not think that the disease shows a progressive tendency in more than one half of the cases.

The various symptoms do not always progress *pari passu*. Sensibility may fail although inco-ordination does not increase. Conversely, the ataxy may become greater although sensation remains the same, and even when it shows a distinct improvement. Thus a patient had slight ataxy, loss to pain on thighs, legs, and trunk, and no loss to touch or temperature. At the end of a year and a half pain was perceived on the thighs, and touch and temperature were still normal,

although the ataxy had increased considerably. So marked indeed is the contrast between the course of different symptoms in some cases that it suggests a certain alternative tendency. If optic nerve atrophy develops, the spinal symptoms, in most cases, remain stationary. So too of visceral crises. I have known gastric crises to cease, when the inco-ordination began to develop. In one patient all the symptoms of tabes passed away, the knee-jerk returned, optic nerve atrophy ceased to advance, but the patient became insane.

When the disease progresses, the rate of its advance varies much. Sometimes it is slow and uniform, and the ataxy is considerable only at the end of several years. On the other hand it may be rapid, so that in a few months the patient is scarcely able to walk. More commonly the progress is varied; periods in which the disease is almost stationary alternate with others in which the symptoms increase rapidly. The exacerbations may seem spontaneous, or may be distinctly excited by some prejudicial influences,—a chill, a fall, or some excess alcoholic or sexual. They are sometimes very acute; a marked change may occur in the course of a few days, or even in a few hours. Thus I have known a patient to pass, in the course of twenty-four hours, from a condition in which he could walk fairly well to one in which he could scarcely stand. Muscular power may lessen in such a sudden exacerbation, or the increase may be confined to the true symptoms of the disease. In the former case it is probable that there is a myelitic or neuritic complication; in the latter there seems to be merely an acute process developed in the nerve-elements in which the chronic disease was in progress. It is often accompanied by acute sensory disturbance. One patient felt for weeks as if his feet were of enormous size, as large as the room he was in. The conditions thus developed may pass away again, leaving the patient on a little lower level than before, or they may persist without any considerable recovery.

There is nothing in the nature of the disease, in most cases, to produce death. The only direct effect of the malady, which has ended life, is laryngeal spasm or paralysis. Even the gastric symptoms, prostrating as they are, are never fatal. Patients who have reached the third stage have lived, bedridden, for twenty years. Death often results from intercurrent maladies, some of which are more dangerous to tabetics than to others because they may develop painlessly and attain a serious degree before their existence is suspected. But many patients die from indirect results of the disease. Kidney complications are the most common; they often develop most insidiously and then, as it were, explode. The patient may be seized with acute febrile symptoms, the exact nature of which may be unsuspected until the urine is examined. I have known a patient, still in the first stage, die thus after a few days' illness. Bedsores and pyæmia occasionally cause death, although less frequently than in some other diseases of the spinal cord.

COMPLICATIONS.—Occurring, as most cases do, in syphilitic subjects, tabes is sometimes complicated by true syphilitic lesions of the brain or cord. Some of the cranial nerve palsies that are observed in the early stage of the disease are of this nature.

Acute or subacute myelitis may occur during the course of the disease as well as before its development. In such cases rapid loss of power, with other symptoms, indicates the occurrence of an acute process involving other elements of the cord than those on which the special symptoms of tabes depend. Such myelitis is probably also, in some cases, of syphilitic origin. Sometimes it is apparently simple; it may develop after exposure to some exciting cause, as in the case already mentioned, in which, after the patient had suffered for four years from gastric crises and pain, complete paraplegia developed a few days after he had sat for some time on wet grass. Such a complication must not be confounded with the simple acute exacerbation in the special symptoms already described.

Other system-diseases of the cord may develop as complications of tabes. The lateral columns may degenerate with the posterior, giving rise to "ataxic paraplegia," but this is rather a distinct affection than a complication of true tabes, and will be so described. Weakness may, however, supervene slowly in the course of locomotor ataxy, without muscular atrophy, and with a persistence of the special symptoms of the disease, from a later extension of the morbid process to the lateral columns. Another occasional complication is progressive muscular atrophy. We have already seen that local atrophy of muscles is not very rare in the later stages of tabes, but in the cases now under consideration, muscular atrophy develops as in its primary form, and runs an independent course, such as it pursues when it exists alone. Thus a gentleman, who had had hemiplegia from syphilitic vascular disease, developed the first stage of tabes—pains, loss of knee-jerk, loss of the iris-reflex, and slight unsteadiness. He had two courses of treatment at Aix-la-Chapelle, and at the end of the second, by which he was much prostrated, the muscles of the hands began to waste. The atrophy spread, and in the course of two years almost all the muscles of the arms, shoulders, and back became extremely wasted. In rare cases the legs present the symptoms of tabes, while in the arms there is the jerky inco-ordination of disseminated sclerosis, and the coexistence of the two diseases has been demonstrated post mortem.

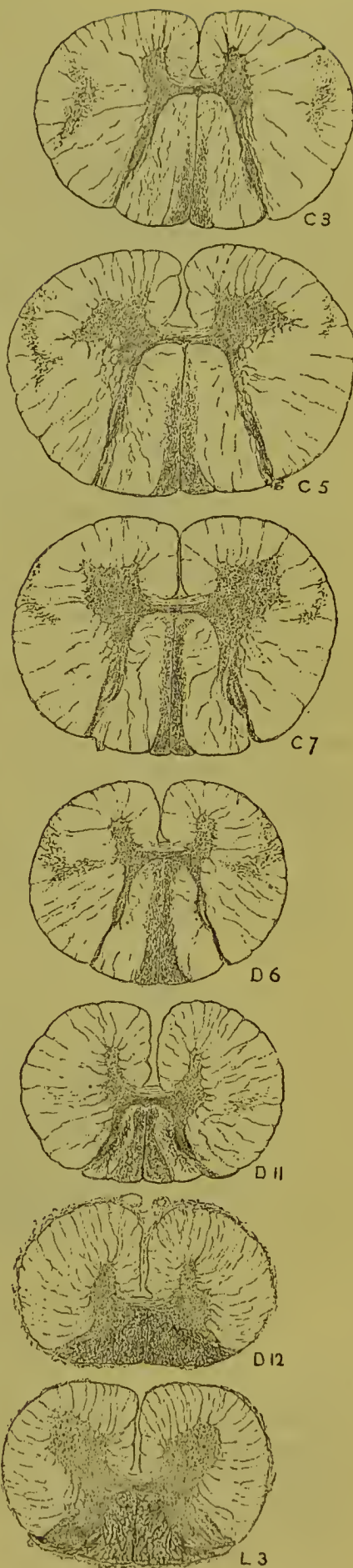
Another very important and frequent complication of tabes is general paralysis of the insane. The two diseases have many alliances. It is probable that syphilis predisposes to general paralysis, as well as to tabes. Reflex iridoplegia is common in both diseases. The two maladies are often combined, and the symptoms of one or the other may preponderate. Thus many general paralytics present symptoms of tabes, and its characteristic lesion is found after death. On

the other hand, cases of tabes may present slight symptoms of general paralysis, perhaps only slight optimism and mental weakness, which may remain subordinate or may increase to a pronounced and preponderant degree. It may be difficult to say in which category a case should be placed. It is sometimes said that a case may commence as ataxy, and may change to general paralysis, but a more correct expression of the facts is the co-existence of the two affections, and the dominance of the symptoms of one or the other.

Of complications of tabes outside the nervous system, valvular heart disease is the most important, and usually develops without any of the usual antecedents of heart disease, and at an age at which degeneration is unlikely. Aortic regurgitation is especially frequent. It is not improbable that the cardiac and neural diseases own a common cause—syphilis, and that the valvular lesion is due to the syphilitic process that is known sometimes to attack the cardiac and especially the aortic valves.

PATHOLOGICAL ANATOMY.—In most cases the spinal cord presents changes visible to the naked eye. The posterior columns have a grey translucent appearance, which is due to the loss of the white myelin of the nerve-fibres

FIG. 111.—Locomotor ataxy involving the legs only. Sclerosis of the whole posterior columns in the lumbar region, gradually becoming limited, in the lower dorsal region, to the root-zone and posterior median column. The latter only is affected in the upper half of the cord (ascending degeneration). There is also ascending degeneration in the antero-lateral ascending tract. This is rather greater on the left side, while the post.-median degeneration is greater on the right, in harmony with the theory put forward on p. 119, that the latter contains fibres which do not decussate.



and to an increase of the connective tissue ("grey degeneration"). The whole of the posterior columns may be thus changed, or only parts of them, the distribution of the visible alteration being that of the histological changes revealed by the microscope and presently to be described. In the cord hardened by bichromate of potash or chromic acid, the difference of tint in the diseased areas is even more distinct than it is in the fresh organ. The posterior columns are also smaller than normal, because the bulk of the connective tissue is less than that of the normal fibres: hence the shape of the cord is slightly changed.

In a section of the cord stained with carmine or other agent, and examined under the microscope, the affected areas are conspicuous by the deep staining of the connective tissue. The position of the excess of this tissue, of the "sclerosis," as it is termed, indicates the place in which the nerve-fibres have degenerated. In the most frequent condition, in which the legs only are affected, the sclerosis occupies the whole of the columns in the lumbar region (Fig. 111) but it is often slight in the anterior parts of the postero-external columns, which may even be free from sclerosis, and in the middle of these columns many normal nerve-fibres may be mingled with tracts of connective tissue. The sclerosis is most dense in the part adjacent to the posterior cornu, through which the posterior root-fibres run, and near the surface of the cord. Above the lumbar enlargement the affection of the postero-external columns gradually ceases, but the degeneration is intense in the postero-median columns, and has the distribution of an ascending degeneration, as it in fact is, receding, in the upper cervical region, from the commissure. (Compare Fig. 111 with Fig. 67, p. 117.) In other cases the external band of sclerosis, adjacent to the cornu,



FIG. 112.—Tabes with ataxy of the arms as well as the legs. Section of cord in cervical region, showing sclerosis of the post.-med. column and root-zone of post.-ext. col. Degeneration of left ant. cornu.

extends up through the dorsal cord and even through the cervical enlargement, blending with the median tract at the periphery (Fig. 112). The median degeneration then extends up to the commissure throughout the cervical region, just as does the secondary

degeneration that results from a cervical lesion (see Fig. 69, p. 118). But in the cervical enlargement the unaffected area in the anterior part of the postero-external column is usually much larger than in the lumbar enlargement.

In rare cases, of severe degree and long duration, the posterior columns are occupied by connective tissue in their whole extent from one end of the cord to the other. A few fibres of the posterior roots and a few vertical fibres near the commissure may alone be recognisable. An instance of such complete sclerosis is shown in Fig. 113.

On the other hand, in slight cases, in which the disease is still in the first, or in the commencement of the second stage, the sclerosis is moderate in degree even in the regions of the cord most affected. The separation of the sclerosed areas in the postero-external and postero-median columns may be distinct throughout the cord, except in the lumbar enlargement, where the median degeneration expands into the external column. The sclerosis of the postero-external column is then usually limited to the root-zone, and varies in width, according to the width of the area of the column through which the root-fibres pass. In the dorsal region there is usually a narrow band of sclerosis close to the posterior cornu; in the lumbar region it reaches almost to the median septum. The anterior part of the post. external column is often free from disease. In some instances, however, a very slight degree of sclerosis extends

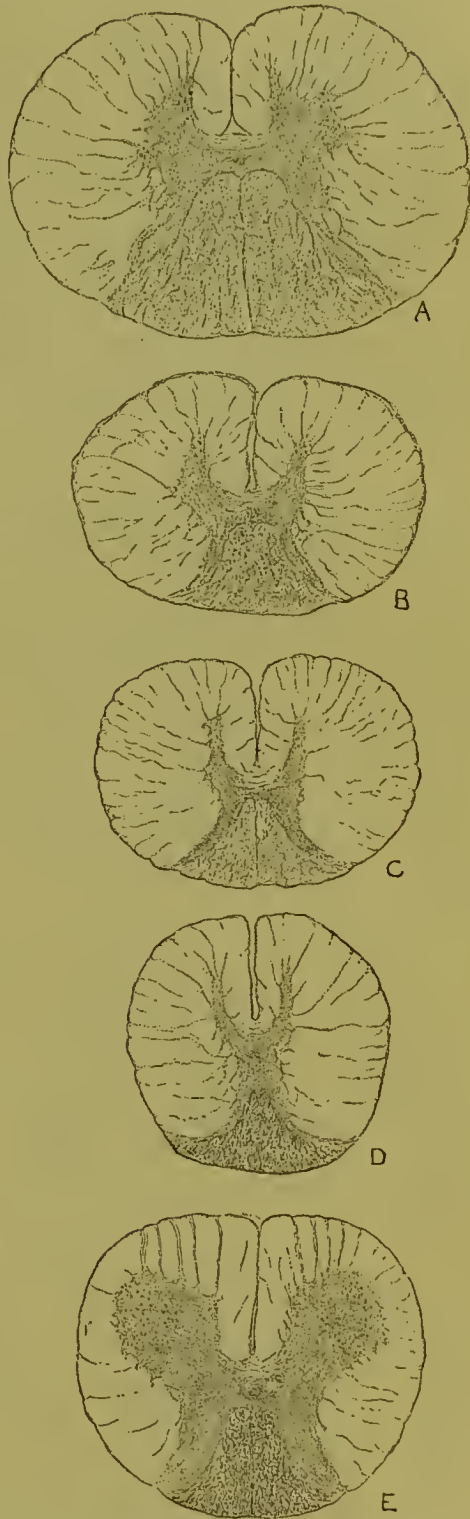


FIG. 113.—Locomotor ataxy: extreme in-co-ordination and anæsthesia in both arms and legs; the posterior columns are sclerosed throughout the cord in their entire extent. A, upper cervical, B, C, D, dorsal, E, lumbar regions. In D there is also some degeneration of the intermediate grey substance.



from the most affected tracts through the rest of the columns and is generally greatest in their posterior half. This condition existed in the very instructive case shown in Fig. 114. The intensification of this diffuse sclerosis in the root-zone and median columns is very distinct, although the affection of the latter is unusually slight. There is, moreover, in the dorsal region, sclerosis of another part of the postero-external column—the comma-shaped tract of short fibres, which degenerates downwards for a few inches below a transverse lesion of the cord (see p. 120 and Figs. 69 and 98, F). It is most distinct at D, 8, but can be traced up to the cervical region, where its form is changed.

The antero-lateral columns may be perfectly normal, even when the

FIG. 114.—Loco. ataxy. Sclerosis of posterior columns. In the lumbar section the posterior portion of both p. m. c. and p. e. c. is densely sclerosed, but in front the disease is limited to the post.-med. col. At the tenth dorsal the sclerosis is also general, but is dense only in the root-zone of the post. ext. col. At the eighth and fourth dorsal the change is slight except in three areas, the root-zone, the middle part of the post.-med. col., and the comma-shaped tract in the front of the post. ext. col. At the first dorsal the comma-shaped tract is narrow and in the lower cervical it merges in a band of sclerosis, which bounds the anterior part of the post.-med. col. There is a little diffuse sclerosis through the rest of the column, and in the first dorsal there is some degeneration of the anterior cornua. Symptoms of the first stage of tabes existed for four years (lightning pains, slight analgesia, loss of knee-jerk, loss of iris-reflex, gastric crises, optic nerve atrophy). A few weeks before death the legs gradually became paralysed, and the arms ataxic; retention of urine caused acute kidney mischief, which was the immediate cause of death. No lesion was found after death to explain the subacute paraplegia.

disease of the posterior columns is great. Often, however, in old cases, there is a slight general increase in the connective tissue throughout the cord. Sometimes there is distinct sclerosis of other definite tracts. The ascending antero-lateral tract may be degenerated above the lower dorsal region, below which it seems not to exist; the fibres probably pass to it from the posterior commissure in the lower dorsal region. An example of this degeneration is shown in Fig. 111. The direct cerebellar tract is also occasionally diseased, but certainly less frequently than has been alleged. In rare cases there is a distinct degeneration of the pyramidal tracts. The peripheral layer of the antero-lateral column is occasionally the seat of sclerosis, the connective tissue extending in from the pia mater; this is usually also thickened, apparently from chronic inflammation, which will be again referred to.

In slight cases it is difficult to distinguish any changes in the grey matter of the cord, although it is probable that there is an atrophy of some of the nerve-cells and fibres in the posterior horn. In more advanced cases such atrophy is constant, as Lockhart Clarke first showed. The posterior commissure may also be smaller than normal. The posterior vesicular column (of Clarke) shows distinct degeneration in many cases. Some atrophy of the plexus of fine fibres contained in these columns is said to be almost invariable (Lissauer). In other cases the change is greater, the cells and vertical fibres waste, and only a few shrunken cells scattered through a translucent nucleated tissue may remain. The atrophy of the cells is said to be associated with sclerosis of the direct cerebellar tract. When the cells are unaffected, although the intervening substance is diseased, the cerebellar tract may be normal, and the fibres passing to it from the front of the vesicular tract may be unaffected. The degeneration of the grey matter often extends into the intermediate region between the cornua (Fig. 113, B); the intermediate lateral tract of cells may be atrophied, and occasionally even the anterior cornu and its cells suffer in a similar way (Figs. 112, 114, D, 1). This change is met with in the cases in which there is secondary muscular atrophy.

The sclerosis varies considerably in its histological characters. In slight cases, there is merely a moderate thickening of the trabeculae and their branches. The larger tracts are more fibrous than normal, and more nuclei are seen than in health. Everywhere the new growth of connective tissue proceeds chiefly from the pre-existing tracts, trabeculae, vessel-walls, &c. When the change is considerable, the trabeculae are very broad, and areas seem to consist wholly of connective tissue, fibrillated and nucleated. In old cases there is sometimes little appearance of fibrillation in the most affected region; cells and thickened vessels lie in granular or homogeneous tissue. From the parts most affected, the nerve-fibres have wholly disappeared. In other parts, however, close examination reveals many fibres narrower than normal, and many points that may possibly be axis-cylinders.

The walls of the vessels are usually increased in thickness, sometimes to a very considerable degree. The coat next the endothelium may be thickened and contain many nuclei, but the chief thickening, in most cases, is in the outer coat and adventitia. The vessels are most affected near the posterior median septum, the posterior surface, and the grey matter. From the thickened wall trabeculae pass off into the adjacent tissue. The pia mater is sometimes thickened over the posterior columns; the vessels in it may be similarly changed. The alteration in

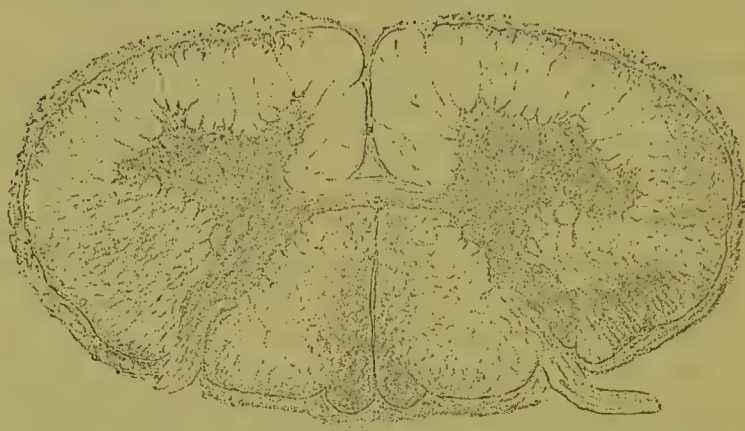


FIG. 115.—Tabes. Chronic meningitis. Cervical region. Sclerosis of post.-median columns and slight diffuse sclerosis of the cord, least in ant. cols., dense in the right lateral column. Thickened pia mater, and damage to subjacent superficial layer of the cord.

the membrane may extend over the neighbouring part of the lateral column, and even around the cord (Fig. 115), and, as already stated, septa passing from it into the cord may be thickened in the circumferential zone. In cases of rapid course, products of degeneration, masses of myelin and granule-corpuscles, may be found in the affected parts.

In rare cases other lesions are found in the cord, corresponding to the complications already described. The lateral columns may be sclerosed apart from any thickening of the membranes. Diffuse myelitis may be met with, and, occasionally, characteristic insular sclerosis has been found in the cervical region with posterior sclerosis in the lumbar.

The posterior nerve-roots may appear normal in slight cases; it must be remembered that only a considerable degree of disease can be detected even by the microscope. Often their disease is conspicuous; when the cord is much affected they are invariably atrophied, grey and thin to the naked eye, and the microscope shows wasting of the nerve-fibres and slight increase of connective tissue. The change may extend down to the ganglia, where it ceases. The ganglia themselves are almost always normal in structure, and the mixed nerve beyond them is free usually from degeneration. The anterior nerve-roots are altogether normal, except in the rare cases in which the anterior cornua have suffered, and then some of their fibres may be degenerated. In this case these atrophied fibres may be traced also in the mixed nerve-trunk beyond the ganglion.

The peripheral spinal nerves have been recently found to undergo atrophy in a large number of cases of tabes.* The change consists in a wasting of the nerve-fibres, beginning in the white substance, which may be reduced to a very narrow layer. Ultimately the axis-cylinders perish. There may be a slight increase in the interstitial tissue and nuclei, but the change seems to commence in the nerve-fibres themselves. The degeneration is greatest in the cutaneous nerve-filaments, and lessens in degree as the nerves are examined higher up. In nerves one or two millimetres thick it is still considerable, although there are more healthy fibres than in the peripheral twigs. In larger nerve-trunks it gradually ceases, and near the spine the nerves are always healthy. The sensory fibres seem to be exclusively affected. The lesion has hitherto been found chiefly in the sensory nerves that supply the skin and joints, but that the sensory nerves of muscle undergo a similar change has been ascertained by Déjerine. The peripheral degeneration is found most commonly in the legs, but is met with also in the arms when these are the seat of symptoms. The change in the nerves bears no relation to that in the spinal cord in degree, or even in existence, for extreme alterations have been found in the nerves when the cord is quite normal. On the other hand, in some cases, the nerves have been examined and found healthy.

The optic nerves when atrophied present wasting of the nerve-fibres, and very often a remarkable increase in the interstitial connective tissue, which forms thick tracts, of gelatinous aspect, between the bundles of nerve-fibres. Degeneration of the ascending root of the fifth nerve has been found (first by Westphal in 1864) in cases in which symptoms were present in the area of its distribution. Sclerosis of the third nerve has also been found.

Pierret has described, as common in cases with vaso-motor and visceral symptoms, a degeneration of the intermedio-lateral tract and adjacent nerve-fibres in the upper part of the cord, traceable up the medulla oblongata in the region of the so-called "slender column," adjacent to the accessory, glossopharyngeal, and pneumogastric nuclei from which it is supposed that the chief visceral and vaso-motor influences are exerted. The sympathetic has been examined in several cases, and found healthy, with one exception, in which the sympathetic on the left side of the neck was degenerated in association with left exophthalmos.

In the cases in which arthritic symptoms have been present during life, or fractures have occurred, conspicuous changes are found after death. There is occasionally an extraordinary wasting of the articular ends of the bones, which seems to begin by rapid erosion of the cartilages, extending quickly to the bone beyond, the extremity of

* The first observation was made by Westphal, but the extent and significance of the change in the nerves was pointed out by Pierret (1880), and his observation was soon after confirmed and extended by Déjerine and Pitres.

which may be much reduced in size and altered in form. In the hip-joint, for instance, in a case recorded by Charcot, the edges of the acetabulum had been removed, and the whole of the head of the femur and most of the trochanter had disappeared (Fig. 116).

Similar changes have been found in most of the larger joints. In the bones, the compact tissue has been found thinner and more porous than normal. At the seat of old fractures a large amount of bony callus is formed (Fig. 116), and sometimes new bone is found in the capsule of joints that have been injured.



FIG. 116.—Osseous lesions in tabes. (After Charcot.)

A. Atrophy of the head of the femur. B. Excessive formation of callus after so-called "spontaneous" fracture of the ulna and radius.

PATHOLOGY.—The great fact of the pathology of tabes is that it is a neural degeneration in the sensory nervous system, peripheral and central. In both peripheral nerves and spinal cord, the incidence of the disease is almost exclusively on sensory structures, and the changes seem to begin in the nerve-elements themselves. The cerebral system suffers in far less degree than the spinal, and in more irregular form, but in the occasional peripheral degeneration of the optic nerve and the central changes in the roots of the fifth, we may trace the same law of distribution. It is less dominant, however, than in the spinal system, since some of the cranial nerve symptoms are exclusively motor. The motor character of the leading symptom, ataxy, is, as we shall see, only an apparent and not a real exception to the general sensory character of the malady.

The degeneration, it has just been said, must be regarded as commencing in the nerve-elements themselves; the overgrowth of connective tissue, which gives to the lesion its obtrusive character, is secondary. That this is true of the spinal cord lesion has, indeed, been doubted, chiefly on the ground of the thickening of the walls of the vessels seen in some cases, and the manner in which the increase of tissue seems to start from them. But the "system" character of a disease, the limitation of a widespread lesion in its early stage or slight degree to structures having a common function, is probably in all cases proof of its neural origin, *i. e.* its origin in the nerve-elements themselves. In tabes the evidence thus afforded is corroborated by the disease of the peripheral nerves, which unquestionably commences in the nerve-fibres, and by the partial character of the sensory loss, which means the limitation of the chief lesion to fibres of one sensory function.

There is every gradation, moreover, between the cases in which the vessels of the cord suffer much, and those in which they are unchanged, and in which there is nothing even to suggest an initial interstitial process. The change in the vessels may reach a high degree in the posterior median columns when their degeneration is secondary, and certainly commences in the nerve-fibres; a similar thickening of the vessels has also been met with in some other instances of purely secondary degeneration. We must, moreover, remember that the overgrowth of the neuroglia is a pathological process of growth distinguishable from the degeneration of the fibres that excites it. It may vary in degree in different cases, and does so conspicuously in secondary degeneration of peripheral nerves, in which it may assume an inflammatory character independent of any propagated inflammation from the primary lesion. Moreover the growth of new is mainly from old tissue, and the chief new tracts are clearly determined in position by the pre-existing tissue. The neighbourhood of vessels seems the place in which connective tissue grows most readily. It is from the trunk that the twigs of a pollard willow spring, but they would not grow there had not the branches been lopped off. Once excited, the energy of tissue-growth may be in some degree independent of its cause. Some vascular disturbance may attend it, and thus a sub-inflammatory condition is probably sometimes developed, which may invade the pia mater, and through this may spread widely. Whether, in some cases, a constitutional state, disposing to tissue overgrowth and tissue inflammation (as syphilis perhaps does in certain stages), may determine the degree of the interstitial change, is a problem of much interest, which does not, however, affect the question which has been just discussed.

The relation of the disease to syphilis is as obscure in nature as it is certain in fact. There is nothing in the histological character of the lesion that can be compared with the morbid processes that are recognised as certainly syphilitic. There is nothing therefore to warrant the opinion that any morbid germs deposited in the cord or nerves lead to the development of the disease. It is probable that syphilis leaves behind it a predisposition to nerve degeneration, lowers, in certain nerve-elements, the capacity for resisting morbid influences, leaves many patients in a condition analogous to that of one who inherits a predisposition to neural diseases. Thus we can understand the length of time that often intervenes between the syphilis and the first symptoms of tabes, the co-operation of other causes, and likewise the fact that other degenerative diseases, besides locomotor ataxy, sometimes occur in those who have had constitutional syphilis.

The pathological facts already known afford an adequate and easy explanation of many of the symptoms of the disease. There may be an interruption of the sensory path in one or both of two places, in the peripheral nerves, and in the posterior root-fibres as they enter the cord, and an interruption of the fibres in either place will explain

the loss of sensibility which is so frequently present. The pains may reasonably be ascribed to the molecular changes of the process of degeneration in the nerve-fibres, either in the periphery or in the cord. The intensity of a sensation is no measure of that of the change that causes it. Whether the affection of the sensory cells of the cord takes part in the production of the pain we cannot tell; such a mechanism is not improbable, since it is certain that degeneration occurs in the posterior horns, and the affection of the antero-lateral ascending tract may be taken as evidence of the degeneration of the cells in which some sensory fibres from the skin terminate.* The loss of reflex action from the skin is explained by the interruption of the sensory path, and an increased irritability of the sensory nerves, the result of the commencing degenerative changes, sufficiently explains both the hyperæsthesia and the increase of reflex action sometimes observed (see p. 147). Whatever theory of the nature of the so-called "tendon-reflex" action is held, the loss of the knee-jerk must be explained by an interruption of the sensory path. On the theory I have advanced, the arrest of impressions from the sensory muscular nerves abolishes the muscle-reflex action on which the local irritability depends. Many other facts of disease show that this irritability is easily lost, and that a very slight change in any part of the reflex arc, too slight to cause other symptoms, is sufficient to arrest the knee-jerk.† Hence we can understand that this loss should be constant and early. It is not improbable, moreover, that these nerves suffer in special degree. The muscles may be insensitive to pain as of electrical stimulation. Pressure and extension, which in health are painful, sometimes cause no sensation, even when the skin is sensitive.

The mechanism of the muscular inco-ordination, which is the obtrusive symptom of the disease, has been the subject of much discussion. Two fundamental facts, however, limit the problem. First, the ataxy cannot be primarily due to the loss of cutaneous sensibility. Disease of the conducting path in the cord may cause absolute anæsthesia of the skin without the least ataxy. Although this does not prove that interruption of the sensory path in the nerves, between the skin and the reflex centres, may not cause inco-ordination, this element seems to be excluded by the fact that there is no relation between the ataxy and the loss of feeling in the skin. There may be, in tabes, much ataxy without any cutaneous anæsthesia, and *vice versa*. The second fact is that ataxy may exist in considerable degree when the lesion is solely one of the peripheral nerves, and the pos-

* In disease of the nerve-roots of the cauda equina there is always ascending degeneration of the posterior median columns, but not of the antero-lateral ascending tract. I have recently had an opportunity of ascertaining this. Hence the root-fibres from which the path is continued by this tract must end in nerve-cells in the posterior cornua, and the degeneration of this tract in tabes is proof of the degeneration of these sensory cells. See the 'Lancet,' June 19, 1886.

† *E.g.* its loss after diphtheria when there are no other symptoms.

terior columns of the cord are free from disease.* These two facts, taken together, seem to show that the ataxy may be produced by one mechanism, even operating alone, the disease of the sensory muscle-nerves. If the loss of the knee-jerk in tabes is to be taken, as I believe it may, as an indication of the disease of these nerves, the constancy of the loss shows the constancy of the presence of this element in some degree. As just stated, a very slight degree of disease may abolish the knee-jerk; it is probable that a considerable degree is needed to cause inco-ordination.

In unilateral lesions of the spinal cord, as we have already seen (p. 157), there may be loss of the sense of posture, with intact cutaneous sensibility, on the side of the lesion, and no loss of this sense on the opposite side, on which cutaneous sensibility is lost. In such a case marked ataxy has been observed on the side on which the sense of posture was lost, when motor power returned.† Whatever effect in causing ataxy is produced by disease of the path of muscular sensibility in the spinal cord must also be produced by interruption of the path between the muscles and the cord. The latter must also arrest whatever reflex action depends upon these muscle-nerves,‡ and it is possible that such reflex action takes some part in the mechanism of muscular co-ordination.

In this connection it is important to note that the fibres that pass up by the posterior median columns probably constitute the path from these nerves. The root-fibres to the columns seem to pass up, as the path is proved to do, without decussating, and they may be diseased in intense degree when there is no loss of cutaneous sensibility. This tract is affected in almost all cases of tabes in which there is a lesion in the cord, and in all such cases in which there is ataxy. This fact suggests that the lesion of the root-fibres is first and chiefly of those from the muscles.§

The posterior median columns (extensively connected with the cere-

* Apart from the evidence of this from cases of true tabes, a very instructive case has been recently recorded by Dr. Hughes Bennett, in which all the symptoms of tabes were present in a case of multiple tumours of the posterior nerve-roots ('Clinical Soc. Transactions,' vol. xviii).

† Gilbert; see footnote, p. 157.

‡ In a case in which the inco-ordination was much greater in the right leg than in the left, and cutaneous sensibility was equally impaired in the two, a strong traction on the calf-muscles, by forcible passive flexion of the foot, produced a distinct sensation in the extended muscles of the left leg, and no sensation in those of the right. In another case, in which the skin was sensitive to the slightest touch, the patient was unconscious of a vigorous extension of the toes produced by faradaic stimulation of their short extensor. This tract is most diseased on the side opposite to that on which there is most degeneration of the antero-lateral ascending tract, which probably conducts sensations of pain.

§ The opinion that the ataxy depends on impairment of the muscular sense was put forward in 1855 by Dr. Russell Reynolds; see p. 288. Déjerine has also associated the ataxy with the degeneration of the peripheral sensory muscle-nerves ('Archives de Physiologie,' 3rd series, vol. iii, p. 231).

bellum) and the direct cerebellar tract probably conduct to the cerebellum the impressions from the sensory muscle-nerves. If so, the disease of these tracts and nerves must interfere with the co-ordinating action of the cerebellum and cause some part of the loss of function which results from disease of the cerebellum itself.

There is another mechanism which may co-operate in increasing ataxy. The vertical fibres of the postero-external column have only a short course, and some probably connect the posterior grey matter at adjacent levels. The affection of these fibres seems to be proved by the interesting fact already mentioned (Fig. 114) that the comma-shaped bundle of fibres in the front of the post.-external column may be specially degenerated. This lesion may impair the association of the sensory structures, and so cause a want of harmony in the central mechanism. The theory that the ataxy was due to this cause was advanced by Todd in 1847. It is not probable that, if effective, it has more than an intensifying influence, since ataxy may exist when the posterior columns are throughout unaffected.

Although cutaneous anæsthesia cannot, alone, produce ataxy it may reasonably be assumed to increase that which already exists. Sensations from the skin furnish important guidance to the motor cerebral centres, and are probably also concerned in such reflex muscular actions as that of standing. Similar guidance to the cerebral co-ordinating centres is also afforded by visual impressions, the loss of which distinctly augments the defect of co-ordination.

The varying characters of the ataxy in different cases probably depend on the local variations in the degree of change in the muscle-nerves. If, as Flechsig believes, the direct cerebellar tract conducts impressions from the trunk-muscles, its disease may produce the trunk-ataxy described on p. 293.*

It is thus probable that co-ordination is chiefly an automatic process, depending partly perhaps on muscle-reflex actions, and on the connection of neighbouring sensory structures in the spinal cord, but chiefly on the function of the cerebellum itself, and the connection of the muscles with it, and that the interruption of this connection is the chief element in the inco-ordination of locomotor ataxy. It is probable also that the automatic processes are in part under cerebral control, guided by sensory impressions which do enter the sphere of consciousness, and that the derangement of this control will intensify inco-ordination, though incapable of producing it. We do not at present know to what extent, in any given case, the symptoms are due to the cord disease or to the peripheral nerve lesion. Apparently pains, ataxy, and anæsthesia may be due to either. The question can only be decided by the comparison of symptoms and pathological changes in a large number

* As already mentioned, the fibres in the posterior vesicular column may be degenerated when the cells suffer but little, and the fibres from this column to the direct cerebellar tract, and this tract itself, are unaffected. Hence the function subserved by this tract may be lost, from the peripheral degeneration of the fibres to the vesicular column, when the tract itself is not diseased.

of cases. That anæsthesia may be due to the peripheral changes is proved by the observed correspondence of the two in distribution (Déjerine).

The trophic changes in the skin, bones, and joints are probably due to the process of degeneration in the peripheral nerves. The degeneration has been found in all cases of the kind in which it has been looked for, and found also in the nerves of diseased joints.

The pains in the region of the fifth nerve are explained by the lesions in its root, especially in its ascending root, which, coming up from the medulla, is homologous with the posterior spinal roots. This nerve, indeed, as Pierret has pointed out, represents the sensory roots of almost all the motor cranial nerves. The degeneration of the optic nerve is fairly comparable with that of the peripheral spinal nerves. Considering the special character of the optic nerve the absence of degeneration of the retina does not seem to destroy the analogy between the two. Regarding the pathology of the visceral crises we know but little. The changes observed by Pierret in the neighbourhood of the centre for the pneumogastric may be indications of the cause of the disturbances that occur within the range of this nerve.

The transient motor symptoms in the limbs, and in the eyeball-muscles are apparently of functional origin. They must be distinguished from the lasting palsies which are probably due to degenerative processes. Sclerosis of the third nerve has been observed, and the loss of function of the internal ocular muscles must be ascribed to degeneration of the related centres in the nucleus of the third nerve. In the account of the diseases of this nerve they are more fully described.

DIAGNOSIS.—The diagnosis of tabes rests on the combination of symptoms already described. In the early stage, the loss of the knee-jerk, together with pains, or unsteadiness on standing with the feet together and the eyes closed, justifies a diagnosis of the commencing affection, provided we can exclude diabetes, diphtheritic palsy, and also a lesion of the anterior cornua or nerve-roots by the absence of wasting of the muscles and change in their irritability. The diagnostic value of the loss of the knee-jerk can hardly be overrated. It is probably never absent in health. If there is doubt as to its loss, the precautions recommended on p. 11 should be adopted. When it is lost and reflex action is in excess, a true reflex movement may sometimes simulate the jerk; the distinction is that the tap sometimes causes a movement and sometimes does not; that an interval, brief but appreciable, elapses before the movement occurs; and that a similar movement is caused by a prick on the skin. In cases in which the knee-jerk is present, the diagnosis of tabes is only justified by distinct and characteristic inco-ordination. In the few cases of this kind that have come under my own observation, the knee-jerk has been either unequal on the two sides, or has been lost on one. Such abnormality is probably the rule in these cases, and the diagnosis is

thus facilitated. In a case with lightning pains, but neither inco-ordination nor loss of knee-jerk, a suspicion of tabes would only be justified by the presence of some other symptom, such as retention or incontinence of urine, loss of sexual power, or loss of the iris-reflex.

The last-named sign is of great diagnostic importance. Its significance is that a degenerative process is at work in the nervous system, and it suggests, therefore, that other symptoms are also due to degeneration. But since the iris-reflex is not always lost in tabes, the negative significance of a normal reflex is far less than is the positive significance of its loss. The practical value of this symptom can hardly be overrated. It puts the observer, so to speak, on the track of nerve degeneration.

There are certain diseases with which tabes is especially liable to be confounded. One of these is multiple alcoholic neuritis. The ordinary form of this disease is readily distinguished by the symmetrical paralysis which is its chief manifestation. In the rare variety of alcoholic nerve degeneration which closely resembles locomotor ataxy in its symptoms, "alcoholic pseudo-tabes" (p. 98), the diagnosis may be very difficult. The disease, moreover, resembles tabes pathologically since the lesion is a "parenchymatous neuritis," subacute degeneration, beginning in the nerve-fibres. It often affects, however, in some degree the motor as well as the sensory nerves, and there is then some weakness in the distal portions of the limbs. An altered electrical reaction may be found in the muscles, and there is generally marked muscular tenderness, scarcely ever present in tabes. The pupils act normally, but this is not an absolute distinction, since they may be unaffected in tabes. A history of alcoholism also helps the diagnosis. By attention to all these points, a diagnosis can generally be made without much difficulty. In rare cases, without motor symptoms, it may be impossible (see p. 100).

When the symptoms and lesion of tabes are combined with those of general paralysis of the insane, it may be doubtful in which category a case should be placed. The question is rather one of the preponderance of the symptoms of one or the other malady than of absolute distinction between them. In most cases, however, in which this combination exists, the symptoms of general paralysis become more pronounced as time goes on, and the spinal symptoms, which at first were the most conspicuous, pass into the background.

All common forms of paraplegia are distinguished by the early loss of power, and by the persistence of the knee-jerk, often emphasised by its excess and by the occurrence of a clonus in the rectus or the calf-muscles. If weakness supervenes in tabes, it is usually late in the course of the disease, and the other symptoms have been and are well marked. If the knee-jerk has been once lost in this disease, it is not reproduced by secondary lesions of the cord which ordinarily increase the knee-jerk. The same distinctions usually suffice for the diagnosis from the combined form

of paralysis and inco-ordination that I have termed "ataxic paraplegia." In this there is always an excessive knee-jerk and a foot-clonus, pains and anæsthesia are absent, and spasm supersedes ataxy. The diagnosis between the two maladies can be better discussed when this disease has been described. In other forms of combined disease of the cord, those, for instance, in which the symptoms of tabes are associated with indications of multiple sclerosis or of progressive muscular atrophy, the diagnosis depends on the recognition of union rather than on the distinction of difference.

Acute lesions of the cord, probably situated in the postero-external column, may produce inco-ordination and pain, but the suddenness of the onset, the limitation of the symptoms, and their tendency to subside, usually suffice for the distinction. Symptoms from this cause are far more common in the arms than in the legs. Ataxy in one limb may also be produced by a tumour growing in the posterior column, but other symptoms soon indicate the tendency of the disease to invade the other elements of the cord. All these cases present the characteristics of a focal lesion with random consequences, rather than of a system-disease with limitation to special function.

In cases of diphtheritic paralysis in which the nature of the sore-throat was not recognised, the loss of the knee-jerk may cause tabes to be suspected, but weakness is usually distinct from the first. The danger of a mistake is greater in the rare cases in which inco-ordination is the chief symptom; and the persistence of ataxy may cause an impression that the malady is really tabes, even if its relation to diphtheria is recognised. The paralysis of accommodation and of the palate which almost always precedes the ataxy, and often passes away as the latter develops, afford sufficient proof of the nature of the spinal symptoms. Pains are absent or trifling and there is usually some weakness in addition to the ataxy. True tabes never follows diphtheria, and the mere sequence, therefore, affords proof of the nature of the case, and assurance that the symptoms will not be permanent, however long they persist.

In the cases in which the dorsal region of the cord is chiefly affected the nature of the disease is often not recognised. The severity of the pains in the trunk may lead to a suspicion of disease of the bones, but the two are distinguished by the special symptoms of each disease and also by the absence in tabes of the increase of pain when the patient moves, characteristic of bone disease, and by the fact that the tabetic pains are wide in distribution and variable in seat, while the pain of bone disease is local and constant. Such cases are sometimes also mistaken for neuralgia of the intercostal nerves, but even if there are no pains in the legs, the knee-jerk is lost. A careful examination will generally reveal some loss of sensibility in the regions in which the pains are felt; and the light reflex of the iris, so commonly lost in tabes, is present in all functional diseases with which tabetic pains may be confounded.

The only organic intracranial disease the symptoms of which present

any likeness to those of tabes is tumour of the cerebellum. In some cases of tabes the unsteadiness bears considerable resemblance to that of cerebellar origin. Loss of the knee-jerk is strongly in favour of tabes; although, in some cases of cerebellar tumour, it is said to be lost, it is much more commonly increased. Anæsthesia and lightning pains are conclusive evidence of the spinal disorder, while in cerebellar tumour severe occipital headache is almost invariable, and considerable optic neuritis is extremely common. Optic nerve atrophy is only secondary to neuritis, and loss of the reflex action of the iris to loss of sight.

The various visceral crises are often mistaken for primary disorders of the deranged organs. Recurring attacks of the character described should always arouse suspicion of their possible tabetic nature, and an examination of the knee-jerk will usually decide the point. Errors in diagnosis are usually due to ignorance of the occurrence of these visceral symptoms.

PROGNOSIS.—The prognosis in tabes corresponds with the facts stated as to the course of the disease. It is not, as was once thought, uniformly bad. Arrest is frequent, considerable improvement is not rare, but perfect recovery scarcely ever takes place. The earlier the stage of the disease, the better is the prognosis. In the first stage the chances are in favour of arrest. When ataxy is developed, the prospect of arrest is less than in the first stage, but is still considerable. One patient came under my care fourteen years ago with distinct inco-ordination, which is no greater now than it was then. Occasionally, even in the typical form of the disease, the amount of improvement is very great. One patient was scarcely able to walk across the room, in consequence of the inco-ordination, when he came under treatment, and at the end of six months his gait was scarcely distinguishable from that of a healthy person. When the third stage is reached the possibility of improvement is far less, but is not quite absent. A woman suffering from pure tabes, when admitted to hospital, had been unable even to stand for six months, on account of the extreme ataxy, but she improved so much that in a few months she was able to walk about the room without help, and has continued, now for six years, to do her household work without difficulty, and even to walk some miles with the aid of a stick. But in the cases that present most improvement all the symptoms of the disease scarcely ever disappear. I have only known in one case the knee-jerk to return, and in this patient the disease had not passed the first stage, although the diagnosis was certain. It is difficult to find any trustworthy indications to guide the prognosis in an individual case. The fact of preceding syphilis influences the prognosis only in one condition, when the symptoms develop rapidly and the patient is seen soon after their onset, and especially when they occur within two years of infection. In some cases of this kind anti-syphilitic treatment does great good. Even in these, improvement cannot be confidently anti-

icipated. I have seen some cases of this character in which no benefit resulted from the treatment. In most cases the only trustworthy guide to prognosis is the observed tendency of the individual case.

Of special symptoms, the pains are often the most distressing, and unfortunately they are the most obstinate symptom of the disease. They may persist in undiminished severity when other symptoms lessen, and on the other hand they may become trifling when the disease increases. Diminution of the pain is a good sign if other symptoms are stationary, but not if they increase. The optic nerve atrophy is usually progressive, but not so uniformly as is often asserted. In a few cases I have known its progress to be permanently arrested. Strange to say, the occurrence of optic nerve atrophy makes the prognosis as to the spinal symptoms better; in most cases of atrophy the spinal lesion remains in the first stage.

TREATMENT.—It is important, in every case, that all influences likely to depress the nervous system should be avoided. Excessive mental work and anxiety are most prejudicial. Physical fatigue is also harmful. In severe and acute cases, absolute rest for a week or two is often useful at the onset of treatment. If the patient walks about, exercise should always stop short of fatigue. Care should be taken to avoid the risk of falls; a severe concussion of the spine will sometimes excite to fresh activity a stationary disease. Exposure to cold is also injurious; a severe exposure may cause acute mischief. If practicable it is well that the winter should be spent in a warm and dry climate. A sea voyage is frequently useful, combining, as it does, a maximum of fresh air with a minimum of exertion. If, however, there is much ataxy, the risk of falls from the motion of the ship must be taken into consideration; it is less than might be anticipated. A sea voyage is especially useful in cases that are already stationary; such patients often return with a diminution in all their symptoms. When the disease is advancing, the influence of a voyage is less frequently distinct. The digestive organs should be kept in careful order; an attack of indigestion or constipation is often attended by lightning pains, which cease when an aperient has acted. Food should therefore be light in quality and easily digestible. The change from an active to an inactive life, which takes often causes, may lead to the development of gout in an individual predisposed to it, either by inheritance or by his previous mode of life, and the tendency must, as far as possible, be obviated by regulation of diet and by frequent aperients. Smoking should be either avoided or limited to a small amount. Alcoholic excess is especially harmful; it sometimes excites a very severe exacerbation of the symptoms, and this is not surprising, since, as we have seen, chronic alcoholism may cause a disease resembling tabes in its symptoms and pathology. Sexual excess is also most injurious. Many patients are preserved from this danger by the disease itself, but it is not always so. Excess seems to have a special

influence on the optic nerve atrophy. Several very painful instances of this have come under my notice. A man in the early stage of tabes, with slight atrophy, some peripheral limitation of the fields, but little impairment of acuity of vision, started on a voyage from Australia to England. The day before he started he married, and the marriage had its common sequel. When he reached this country he was quite blind.

Drugs have certainly some influence on the disease, but it is rarely sufficient to check the morbid tendency when this is very active. Moreover, the influence of drugs is variable; that which seems to do much good in one case has no influence on another, and this is true also of the same case at different periods. Hence the therapeutic statements that have been made by various observers are somewhat discordant. The first question in the treatment of any disease that is related to syphilis, is the influence of anti-syphilitic agents. In tabes such influence is distinct only in rare cases, chiefly under the conditions mentioned in the section on prognosis. In those circumstances, full doses of iodide of potassium should be given, and, if the interval since the primary disease is short, mercury may be rubbed in until the gums are slightly affected. In cases of slow development, several years after primary syphilis, iodide seldom does good. Still, in rare cases, improvement follows its administration, and therefore (as well as to clear the therapeutic ground) it is desirable to let the patient take iodide of potassium, if he has not already done so, in doses not exceeding thirty grains a day, for six or eight weeks. Very small doses of mercury, such as $\frac{1}{16}$ th grain of the red iodide, may also be given for a longer time, and may often be conveniently combined with other drugs. It seems sometimes to have a tonic influence, and perhaps may lessen the secondary subinflammatory process in the cord and pia mater, which, as we have seen, sometimes constitutes part of the morbid process. But in such late cases, and in all the late nerve degenerations that follow syphilis, energetic mercurial treatment only does harm. Its depressing influence often increases the degenerative tendency, and hastens the progress of the disease. I have seen several such cases in which an energetic course of mercurial treatment, at Aix-la-Chapelle for instance, had an effect which was simply disastrous.

Of drugs that have been recommended for tabes, those that are most useful, in the order of their value, according my own experience, are arsenic, iron, quinine, nux vomica, nitrate of silver, Calabar bean, belladonna, ergot, phosphorus. Of these arsenic is certainly that which most frequently does distinct good. Most of the cases in which I have known the greatest improvement to occur have been taking it at the time. In several of these cases there was no other change in the conditions of life to which the improvement could be ascribed. The form in which arsenic is given matters little; $\text{m}\nu$ or $\text{m}\chi$ of the Liq. Arsenicalis may be given in mixture, or $\frac{1}{12}$ th gr. of arseniate of soda in a pill. Iodide of arsenic has no special advantage, and is

not so well borne. This or any other drug must be continued for at least two months before an opinion of its influence can be formed. Rapid improvement can never be expected in so chronic a disease. In connection with the influence of arsenic, it is interesting to note the well-known effect of the drug in improving the nutrition of the skin, and the frequency with which the cutaneous nerves are degenerated in tabes. Quinine, nux vomica, or iron may be conveniently combined with arsenic. Nitrate of silver is far less useful, but it may sometimes be given with advantage alternately with arsenic. It should not be given in larger quantity than half a grain a day, nor continued for a longer period at a time than two or three months. A grain a day has been said to cause fatal kidney disease, and I have seen a case in which a still smaller dose taken daily for a long time caused both wrist-drop and albuminuria.

Calabar bean has been recommended by Ringer as producing temporary improvement in many chronic spinal diseases. Its influence is not great, but it seems to do no harm. Ergot is rarely useful in chronic diseases of the cord, and it is difficult to understand the estimation in which it has been held in the treatment of tabes. It may perhaps have some influence on the dilated vessels that are to be seen in many cases, and may therefore be given in the cases in which spinal pain suggests congestion of the membranes, or in which an acute exacerbation of the symptoms, or their extension beyond the special tabetic limits, makes it probable that there is a secondary subinflammatory process. Some years ago I gave gold a careful trial, but without obtaining any satisfactory results.

Counter-irritation to the spine, by blisters or the actual cautery, is sometimes useful, chiefly in cases just mentioned, in which the disease develops rapidly, or the symptoms rapidly increase, or in which there is spinal pain or tenderness. It is not probable that it influences the process of degeneration, but it may lessen any secondary inflammation.

Of special symptoms that require treatment the pains are most important. Unfortunately they are often most obstinate, and their variability renders it very difficult to estimate the influence of treatment upon them. Severe paroxysms may yield only to hypodermic injections of morphia, but this should be reserved for the most intense attacks, since its use is undesirable for a recurring symptom which may continue for years. Among other sedatives, Indian hemp is often effective; gr. $\frac{1}{4}$ or $\frac{1}{2}$ should be given every three or four hours during a paroxysm. Other sedatives are of little use. Hypodermic injections of cocain are uncertain in their influence on tabetic pains, but sometimes relieve the local pain that is accompanied with hyperæsthesia. The only external application that is of service is chloroform sprinkled on spongio-piline (or lint and oiled silk), but this relieves only the superficial pain. Occasionally the pains are lessened by a warm bath. Counter-irritation to the spine, as by repeated sinapisms, sometimes gives some relief, but probably only when the pain has a spinal origin.

Visceral symptoms rarely yield to special treatment; when severe morphia alone mitigates their intensity. Slight gastric crises may be relieved by simultaneous sinapisms to the epigastrium and neck. Laryngeal spasm is usually at once relieved by nitrite of amyl; and the local application of cocain is also useful. For weakness of the bladder, nux vomica, or strychnia is the most useful. The latter may be given by hypodermic injection, as recommended in the chapter on muscular atrophy. Nocturnal passage of urine is often stopped by belladonna.

If blisters or sores form on the feet they should receive careful local treatment until they are well. If neglected they may become very troublesome. Tabetic patients should be cautioned never to cut a corn; perforating ulcer is often set up by a corn being cut too deeply. The epidermis should be softened with an alkali and rubbed off with pumice stone.

Attention to the bladder is one of the most important elements in the treatment of tabes, as in that of all diseases of the spinal cord, chronic and acute. If there is any reason to believe that the bladder is imperfectly emptied, a catheter should be passed, and if residual urine is found, the bladder should be emptied perfectly and washed out every day or every other day. No hesitation need be felt in the use of the catheter in these cases. Scores of patients die every year from want of the catheter because it is thought that all is right if they pass urine freely. The residual accumulation or the slight cystitis sets up chronic pyelo-nephritis, which develops insidiously, and is unsuspected until mysterious febrile disturbance occurs, chronic or acute, and leads to death. It is probable that the onset of the final acute disturbance is sometimes determined by the passage of a catheter, just as this will cause a rigor or transient hæmaturia in a healthy person, and then the case is called "catheter fever." I have seen many melancholy instances of death from this cause, which might have been prevented, and I have never seen any case in which the early and frequent use of the catheter did harm. It is especially important to insist on the danger that the practitioner may be misled by the patient, who thinks that because he passes urine he necessarily empties his bladder.

Electricity has little influence on the chief symptoms of tabes. The voltaic current is powerless over either the pains or the ataxy, whether it be applied to the spine or the limbs. Faradisation of the skin by the wire brush has been recommended for the defect of cutaneous sensibility, but it has little if any influence. When the bladder or its sphincter is weak, faradisation from the hypogastrium to the perineum may be employed, and occasionally seems to do some good. In secondary wasting of the muscles, an attempt may be made to maintain their nutrition by stimulating them with whatever current they respond to, but the central cause usually renders local treatment ineffective.

There remains for consideration one therapeutic procedure,—nerve

stretching, an operation that has usually been performed on the sciatic nerve. In the first case in which it was employed (by Langenbuch), the procedure was followed by remarkable and mysterious improvement, not only in the pains, but also in the inco-ordination. The patient subsequently died under chloroform, administered in order that the nerves of the arms might be stretched. The spinal cord was found by Westphal to be healthy. Although the nerves were not examined, there can be little doubt that it was one of the cases in which the nerves alone are diseased. The operation has since been performed in a large number of cases. In some the pains have been lessened for a time; in a few there has been some temporary improvement in the inco-ordination. In many cases the procedure has had no influence on the symptoms. If ever justified it is only as a last resort, in a case in which the pains are very intense, and are especially felt in the region of the sciatic, but it is not justifiable in any case to hold out an expectation of more than possible, and perhaps transient, relief to the one symptom. It must be remembered, moreover, that the operation is not devoid of danger of evil results; there is the risk incidental to the necessary anæsthetic, and the operation has also caused death through the agencies of erysipelas and spinal hæmorrhage. Its *modus operandi* is not easy to explain, and the theories that have been advanced to account for its influence are so inadequate as to be scarcely worth reproduction. Its common inutility is more easily intelligible. It would seem now to be passing into merited disuse.

PRIMARY SPASTIC PARAPLEGIA

(PRIMARY LATERAL SCLEROSIS).

The morbid state thus designated is one that has been, and still is, the subject of much discussion. Its relations are complex, and a somewhat lengthy pathological introduction is necessary.

We have seen that, in every kind of transverse lesion of the spinal cord, provided this is situated above the lumbar enlargement, the paralysis of the legs is soon accompanied by excess of myotatic irritability (increased knee-jerk, foot clonus), and that the muscle-reflex action, on which this irritability (and muscular tone) seems to depend, gradually increases to tonic spasm, so that a condition develops to which the term "spastic paraplegia" is applied. A condition, quite similar to that which is thus secondary to a transverse lesion, often develops gradually, without any indication of a primary focal disease, and without any sensory symptoms to indicate that the mischief

extends beyond the purely motor elements of the cord. We have also seen that these symptoms indicate disease of the upper segment of the motor path, the cortico-spinal segment, which extends from the motor cortex, through the pyramidal tracts, and ends in the grey matter of the cord, doubtless by a subdivision and ramification of the nerve-fibres in the fibrillary network of the spongy substance. From the gradual onset and limitation of the symptoms in these cases, it has been assumed that the disease consists of a primary sclerosis of the pyramidal tracts, *i.e.* in a degeneration of the fibres of this upper segment. Since these tracts run chiefly in the lateral columns, the disease has been termed "lateral sclerosis." The clinical features presented by these cases, and their probable significance, were first pointed out by Erb.* Degeneration of the pyramidal tracts had indeed been previously observed, by Türck (1856) and Charcot (1865). Erb's inference as to the nature of these cases was supported by the independent (and indeed previous) researches of Charcot on cases of muscular atrophy; he showed that in such cases muscular rigidity coincides with degeneration of the pyramidal tracts. Pathologists have since been searching for confirmation of the hypothesis—for evidence that the symptoms, in their pure form, without muscular atrophy, depend on degeneration limited to the pyramidal tracts. Such degeneration, in slight degree, associated with slight symptoms, has been found in cases of general paralysis of the insane (by Westphal and others), but in all other cases that have been examined, either other parts of the white substance have been degenerated, or the disease has involved also the anterior cornua.† In some cases, as one published by Dreschfeld (of which a figure is given at another page) the change in the anterior cornua has been slight, so that the required conditions are nearly but not quite fulfilled.

Although absolute demonstration has not yet been furnished, the indirect evidence of the correctness of Erb's hypothesis is very strong, the only question being whether, in the cases in which there is no disease of the motor nerve-cells of the anterior cornu, the degeneration is so limited to the pyramidal tracts as to constitute a system-disease in the strict sense of the word. It is not surprising that demonstration of the nature of the pure cases is not forthcoming, since, as we shall see, the disease has very little tendency to shorten life.

Cases that present the same group of symptoms are very various in nature. We have seen (p. 144) that, in each segment of the motor path, the same symptoms are produced by disease of any part of the segment. In the upper segment, with which we are now concerned, the symptoms are the same, whether the disease is in the cortex of the brain, the

* In 1875, 'Berlin klin. Wochenschr.,' No. 26; 1877, 'Virchow's Archiv,' Bd. 70.

† In a case recorded by Stofella no other part than the pyramidal tracts could be seen diseased on naked-eye observation, but no microscopical examination was made.

internal capsule, the pyramids of the medulla, or the pyramidal tracts of the cord; and they must also be the same if the disease is limited to the termination of the segment in the grey matter of the cord. In cerebral hemiplegia the state of the arm may closely resemble that present when spastic paraplegia involves the arm. The leg in hemiplegia presents a less close resemblance to its condition in spinal disease, because the leg is innervated from both cerebral hemispheres, and the supplementary influence of the hemisphere of the same side lessens the effect of the disease of the hemisphere of the opposite side. But if there is disease of the leg centres in both hemispheres, the state of the legs may be identical with that resulting from disease of the spinal cord. Such bilateral disease often results from injury during birth,—meningeal hæmorrhage over the upper part of the central convolutions. This resulting condition is termed “congenital spastic paraplegia.”*

On the other hand, it is extremely probable that degeneration is sometimes limited to the termination of the segment in the grey matter. This is probably susceptible of isolated disease, just as the corresponding termination of the lower segment is paralysed alone by curara. In such disease the white columns would be found normal. It is highly probable that this was the pathological condition in one recorded case, in which the symptoms of spastic paraplegia existed during life, and no anatomical change was discovered after death. The detection of the disease of the terminal portion of the segment in the grey matter is extremely difficult, perhaps impossible, because the structure must consist of nerve-fibrillæ that interlace with others that are unaffected. In other cases, some degeneration has been found in the lateral pyramidal tract in the lumbar region, slighter, however, than the intensity of the symptoms suggested, and gradually lessening or even ceasing higher up the cord. Such a condition is comparable to the degeneration of the lower portion of the second segment of the motor tract in some cases of peripheral (degenerative) neuritis, in which there is a degeneration of motor nerve-fibres, greatest in their extremities, extending for a variable distance up the nerves, but lessening long before the spine is reached.

Both clinical and pathological evidence shows that the morbid state often occurs also as part of a more extensive degeneration. Degeneration may occur in both lateral and posterior columns, giving rise to combined paralysis and ataxy, “ataxic paraplegia,” sufficiently definite in its clinical characters and course to make its distinction convenient, and it is therefore separately described. The following account of the clinical features of spastic paraplegia is founded on cases in which the motor paralysis and spasm existed alone, with no definite sensory loss, in which they came on gradually, with nothing in the state of the patient

* Attention was called to these infantile cases by v. Heine as early as 1840, by Little, in his work on ‘Deformities,’ in 1853, and in the ‘Obstetrical Transactions’ for 1862. See McNutt, ‘Am. Journal of Medical Science,’ January, 1885.

or the history of his symptoms to suggest either a focal lesion or an acute process.

CAUSES.—An inherited neuropathic tendency is to be traced occasionally in this as in other chronic spinal diseases. Thus in one case there was a history of insanity in uncle, aunt, and two cousins. The disease affects both sexes in almost equal frequency, presenting in this a contrast to posterior sclerosis. The period of life at which it most frequently begins is between twenty and forty; about three quarters of the cases begin in these two decades, and about an equal number in each. Cases frequently commence, however, in the second decade of life, chiefly in its latter portion. After forty they become much less common; the latest age at which I have known a typical case to commence is sixty-one.

The disease sometimes follows syphilis in a way to suggest a causal relation, even when all cases are excluded in which there is any reason to suspect a focal lesion. In one case the symptoms commenced six months after the primary disease.* But this antecedent is not frequent, and in this respect also the disease contrasts with posterior sclerosis. Proximate causes are to be traced only in a minority of the cases. The most frequent is concussion of the spine, such as a fall on the back. Some time, often two or three years, elapses between the fall and the first pronounced symptoms of the disease, and hence focal lesions due to the fall can be excluded. Next in frequency is repeated exposure to wet cold. Very rarely the symptoms have slowly followed some acute illness. In several cases the disease has succeeded prostration after childbirth or abortion, or has commenced during lactation. In one case an attack of arthritis in the knee and ankle of one leg was the immediate antecedent, and this leg was the first to become weak. In another case the symptoms followed sub-acute arthritis of both knee-joints, apparently rheumatic in nature. The possibility that a joint inflammation may be of spinal origin must be borne in mind in considering the significance of such cases, but it is on the whole probable that a primary joint affection is an occasional cause of the spinal disease.† Acute arthritis, certainly of spinal origin, has only been observed in severe myelitis. The cause of the congenital form is always injury to the brain during birth, in most cases meningeal hæmorrhage, perhaps sometimes compression of the cortex.

SYMPTOMS.—Weakness of the legs, of very gradual development, is the first symptom. The patient finds that he gets tired more readily than before, and that the legs feel heavy. It is very common for one leg to become weak before the other. The progress of the weakness is very variable, but it is slow in all characteristic cases. In many instances the patient is still able to walk a mile or two, even after

* The cord lesion was found to be sclerosis, but the direct cerebellar tract was also degenerated (Minkowski).

† See 'Arthritic Muscular Atrophy.'

the disease has lasted for several years, slowly increasing. On the other hand, walking power may be almost lost at the end of six months. It is doubtful whether more acute cases should be classed in this category. When the patient seeks advice, the degree of weakness is thus very variable. It will generally be found that there is very distinct loss of power in the flexors, often greatest in the flexors of the hip, but considerable also in those of the knee and ankle. The knee-jerk is excessive and quick; the rectus contraction can be obtained, as the patient lies, by tapping the depressed patella, and a rectus-clonus is often obtainable by sudden depression of the patella. The foot-clonus is usually also obtained with readiness. In rare cases, in which the upper part of the legs suffers most, there may be a rectus-clonus, but only slight indications of a foot-clonus, two or three movements quickly ceasing. The tendency to spasm is at first noticeable as slight stiffness of the legs on first rising in the morning, but it gradually increases in degree as power lessens, until at last the legs, whenever extended, pass into a condition of strong extensor spasm, rigidly fixing them to the pelvis, so that, as the patient lies, if one leg is lifted from the couch by the observer, the other leg is moved also. The spasm may be such that the knee cannot be passively flexed by any force that can be applied to it, until the spasm has become less. When flexed the limb is comparatively supple, but if it is then extended, the spasm instantly returns, making the limb rigid, and often completing the extension, just as the blade of a knife opens out under the influence of its spring, "clasp-knife rigidity." The spasm is, roughly speaking, proportioned to the loss of power, and its extensor character may enable the patient to stand, the legs being fixed and rigid, when muscular power is quite insufficient to support the body, were it unaided by the spasm. In a still greater degree, a voluntary effort may only excite general spasm of the limb instead of causing a definite movement. Occasionally there are also paroxysms of brief flexor spasm, causing the legs to draw up. This occurs chiefly when the patient is in bed, and especially during sleep. When the spasm is great, a paroxysm may pass into violent clonic spasm. This was termed "spinal epilepsy" by Brown-Séquard, but it has, of course, nothing to do with epilepsy. It is merely the same clonus that can be produced by passive extension, excited by the tension of the spontaneous spasm. A paroxysm can often be arrested by a strong sensory impression, such as a prick or a pinch, and also, as Brown-Séquard has shown, by a forcible flexion of the great toe, which probably acts in a similar manner. Reflex action from the skin is also usually excessive, and the stimulus excites an attack of spasm.

The gait of the sufferers from spastic paraplegia is very characteristic. The legs seem to drag behind the patient, and, in walking, each is hauled forwards as a rigid whole, the toes catching against the ground, and, when the ball of the foot rests on the ground, the limb

may shake from the clonus developed by the extension of the calf-muscles. When the patient sits, a similar trepidation occurs until the patient pushes the leg forwards so that the heel rests also on the floor. The muscles of the legs are usually large and well nourished. They often seem indeed to be hypertrophied, and perhaps are really so; the contraction of spasm constitutes a physiological stimulus to growth just as does voluntary effort. But they are not always large; they are sometimes moderately wasted. Rarely they are much wasted; such cases are considered in the chapter on muscular atrophy. The electric irritability is usually perfectly normal, but in the muscles that present the slight wasting just mentioned, there may be a trifling diminution of irritability, to faradism and voltaism alike. When the patient ceases, or almost ceases to walk, contracture sometimes occurs in the calf-muscles. An active contracture is indeed common, preventing at first passive flexion of the ankle beyond a right angle, but if the pressure is maintained, the muscles yield and full flexion is possible. But in some cases there is a fixed contracture that cannot be overcome. This rarely develops while the patient is walking about, but is common when rest in bed permits habitual extension of the foot on the leg. It occurs especially when there is slight muscular wasting. Flexor contraction at the hip or knee may occur also in the same cases, under the influence of posture, but is on the whole rare, the tendency to it being counteracted by the extensor spasm.

The arms are often unaffected, but they suffer in some cases, and present the same progressive weakness and excess of myotatic irritability. Vigorous contractions are elicited by a tap on the tendon of a muscle, or on the bone to which the tendon is attached, provided the muscle is extended. A clonus can often be obtained in the flexors of the fingers. There is less paroxysmal spasm than in the leg, but much rigidity of the limb, from active muscular contracture, flexing the elbow and flexing the fingers at all the joints, as in the "late rigidity" of hemiplegia. The difference in the form of spasm in the arm and leg is no doubt connected with existence of the reflex extensor mechanism, in the lumbar centres, concerned in the act of standing. The muscles of the arm are often as well nourished as those of the leg. The affection of the two arms is very rarely equal, and it is common for one arm to be almost normal when the other is much affected, and both legs are equally involved. Sometimes, however, the arm and leg on one side are paralysed, and the limbs on the other side very slightly. In comparison with the palsied members those on the other side may seem normal, and the patient often believes that they are, but examination almost always reveals slight weakness and an abnormal degree of myotatic irritability.

The muscles of the trunk may also suffer. A subjective "feeling of weakness" in the back is often complained of early, but seems to be as much a sensory as a motor symptom. Painful spasm of the abdominal muscles may occur in severe cases, and I have even known

attacks of tetanoid rigidity of the back to occur on attempts to move.

It is not common, in typical cases, for the symptoms to extend into the region of the cranial nerves, but in rare instances difficulty of swallowing and of articulation has existed, due to a similar palsy of the bulbar nerves. Excessive myotatic irritability has also been observed in the muscles of mastication, so that a tap on the chin causes a vigorous elevation of the depressed jaw (Beever; see p. 150).

Sensory symptoms are often entirely absent, with the exception of slight dull pains in the legs, or more frequently in the back. The sensation of weakness in the back, already mentioned, occasionally reaches an intense degree. Rheumatoid pains are troublesome in some cases; sharp pain is rare, except in untypical cases to be presently mentioned. In such cases also there may be very slight defect of sensibility, such, for instance, as blunting of tactile sensibility on the finger-tips. Subjective sensations of "numbness," tingling, formication, are more common. They may exist for years without the development of any other anæsthesia, in spite of the progress of the motor symptoms.

The sphincters are often affected, and sometimes very early in the course of the disease. Sexual power may be lost, or may remain, even when the spastic paraplegia has reached a high degree. The nutrition of the skin and joints undergoes no change.

Ocular symptoms are extremely rare. The action of the pupils is usually perfect, and I have only once seen optic nerve atrophy in an uncomplicated case.

The infantile form may resemble very closely that which occurs in adults. There is the same extensor spasm and increase of all forms of reflex action. As the child sits on the knee or a chair any sensory stimulus will make the legs shoot out in spasm (Fig. 117). But the spasm does not reach the extreme degree often attained in the common



FIG. 117.—Congenital spastic paralysis (cerebral). Extensor spasm in the legs excited by a sensory impression.*

form. The excess of the knee-jerk is always distinct, but a foot-clonus is not often to be obtained, perhaps because the muscle-reflex mechanism related to the calf-muscles has not received the functional development

* Drawn by Dr. H. R. Spencer, from a photograph by Mr. Hyde Marriott.

that must result from the ever-recurring sequence of tension and contraction involved in walking. The active contracture in the calf-



FIG. 118.—Infantile spastic paralysis of cerebral origin: crossed-legged progression. (Drawn by Dr. Spencer, from a photograph.)

muscles, which most cases present, is a serious hindrance to walking even when the muscular power is sufficient. It is long before the attempt to walk overcomes the contracture. In most cases, however, the child ultimately gains the power of walking, although much later than normal, and it often presents some peculiarity of gait, sometimes a tendency to "cross-legged progression" in which one foot gets over or in front of the other (Fig. 118), or a swinging oscillation of the body occurs which may persist to adult life. The growth of the legs is hindered.

The arms do not present tonic spasm such as is seen in adult cases. There may be a choreoid disorder of movement, spontaneous irregular movements with inco-ordination. In the cases that can fairly be called "spastic paraplegia," the arm symptoms are slight. When considerable, the condition is usually termed "double athetosis," and its characters are described in the account of diseases of the brain.

Transitional forms are met with which constitute gradations between primary spastic paraplegia and other varieties of sclerosis of the spinal cord. Indications of posterior sclerosis may coexist, and to the motor symptoms there is added inco-ordination, sometimes with other sensory symptoms. These cases of "ataxic paraplegia" are described in the next section. Cases may begin as pure tabes, and indications of lateral sclerosis may be superadded. On the other hand, muscular wasting in the arms, considerable in degree and progressive in character, may be associated with indications of lateral sclerosis in the legs, cases to which the name "amyotrophic lateral sclerosis" has been given by Charcot. Very rarely considerable muscular wasting succeeds, in the same part, the symptoms we have been considering.

Lateral sclerosis of the spinal cord (like posterior sclerosis) often forms part of the morbid process that constitutes general paralysis of the insane. In some cases of this character the mental symptoms are extremely slight, and the case may have the aspect of a primary spinal disease, with slight mental failure complicating it.

The course of characteristic cases of primary spastic paraplegia is usually very chronic, and the malady may, at any stage, cease to advance. In one case slight symptoms have apparently been stationary now for twenty years. Often, however, the disease reaches a considerable degree before it becomes stationary. It is perhaps the least dangerous to life of any chronic spinal disease. Even secondary

kidney trouble scarcely ever occurs; perhaps the excessive reflex action may save the bladder from injurious over-distension. It is when other elements of the cord suffer that dangerous complications ensue.

PATHOLOGICAL ANATOMY.—In the fact just stated we probably have an explanation of the silence of morbid anatomy on the subject of uncomplicated lateral sclerosis. "*Nec silet mors*," the apt motto of the Pathological Society, is true chiefly of disease that kills. Complete degeneration of the pyramidal tracts, anterior as well as lateral, is met with in the cases in which the anterior ganglion cells and motor nerves are also diseased, although, in some instances, the amount of this disease is small, and limited to the cervical region. An instance of such degeneration is shown in Fig. 119. The degeneration in the lumbar enlargement is usually limited to the lateral tract, the anterior having disappeared higher up the cord. It is bounded externally, in the dorsal and cervical regions, by the normal direct cerebellar tract, and internally by the narrow "lateral limiting zone," which intervenes between the pyramidal tract and the intermediate grey matter. There is the usual increase of connective tissue and wasting of nerve-fibres. In many cases granule cells are abundant in the affected area. They are always present in cases of short duration and indicate the stage rather than the nature of the process. The degeneration probably begins in all cases in the nerve-elements themselves. In most cases many nerve-fibres can still be seen, scattered through the sclerosed area; most of them are fibres of the direct cerebellar tract



FIG. 119.—Sclerosis of the lateral and (in the cervical region anterior) pyramidal tracts, with slight degeneration of the anterior cornua. A. Cervical; B, dorsal; C, lumbar sections.*

* I am indebted, for these sections, to Dr. Dreschfeld, who has published the case ('British Med. Journal,' Jan. 29th, 1881).

which lie among the pyramidal fibres (see p. 121). The degeneration of the tract has been traced through the medulla, pons, and cerebral hemispheres, to the motor cortex, in which indications of degeneration have also been found. This degeneration through the brain was traced in one case in which the disease of the anterior cornua was slight, the wasting was limited to the hand-muscles, and the spastic paralysis began in the legs and then invaded the arms.* Such a case is almost a pure degeneration of the whole of the first segment of the motor path. The degeneration of the pyramidal tracts is well shown in Fig. 120, from a case of progressive muscular atrophy. The anterior and lateral tracts are entirely degenerated, and the sclerosis stops abruptly at the outer margin of the lateral, but in front extends forwards, probably in the short fibres that connect the anterior cornu



FIG. 120.—Sclerosis of the pyramidal tracts, lateral and anterior: dorsal region. From a case of muscular atrophy.

at different levels. In other cases, in which the anterior ganglion cells were normal, the sclerosis has not been limited to the pyramidal tracts; the anterior columns have been also sclerosed in the lumbar region of the cord.† The direct cerebellar tract (and posterior vesicular columns) have been also found degenerated with the pyramidal tract, and an annular sclerosis of the periphery of the antero-lateral column has been found in several cases.‡ More frequent

* Kojewnikoff, 'Arch. de Neurologie,' 1883, No. 18.

† As in a case recorded as one of spastic paraplegia by Hopkins ('Brain,' Oct., 1883), but this was an example of combined lateral and posterior sclerosis (ataxic paraplegia). An illustration of the changes in this case is given in the next section.

‡ Direct cerebellar and pyramidal tracts in a case by Minkowski, in which the disease rapidly succeeded syphilis; annular sclerosis by Westphal (see "Ataxic Paraplegia").

still is a combination with degeneration of the posterior columns, described more fully in the next section. In most of these combined cases, the degeneration has lessened in the upper part of the cord. In one case, at least, the white columns of the cord have been found healthy. The probable significance of this fact, and the general conclusion from the anatomical evidence, has been already stated in the introductory paragraph. One point, however, that deserves mention is the occasional occurrence of lateral sclerosis in association with insular sclerosis. In some cases of this character the degeneration of the pyramidal tracts is purely secondary, the result of the damage to the pyramidal fibres by an islet of sclerosis situated in some part of their course. But it does not appear that this explanation can be given of all cases. In some instances the sclerosis of the pyramidal tract appears to be independent and coincident. A similar association of insular and posterior sclerosis has also been observed.

The probable mechanism of the symptoms that give the dominant characters to the malady, have been considered in the general account of the symptoms of disease of the spinal cord. The most important fact is that the degeneration, whatever its upward extent, always involves the lowest part of the pyramidal segment, in consequence of its descending tendency. Hence the intra-cornual termination must always be involved. This, it is assumed, is the structure that controls the muscle-reflex centre, and the consequent loss of control explains the excess of myotatic irritability and the progressive spasm, progressive by a functional hypertrophy resulting from continuous over-action. When there is the rare consecutive wasting of muscles, we must conceive that the degeneration involves also the ganglion cells and lower segment of the motor path; when, as is the rule, there is coincident wasting of other muscles than those that are the seat of spasm, it would seem that some elements of this lower segment are the seat of a primary degeneration. The slighter muscular wasting without, or with only trifling, change in electrical irritability, is the expression of a slighter alteration in the nutrition of the cells and fibres, without actual destructive degeneration.

DIAGNOSIS.—The diagnosis rests on the combination of weakness, excess of myotatic irritability, and spasm,—on the gradual onset of these symptoms,—and also on the absence of indications of a focal lesion. An acute onset, occupying a few days or weeks, is *primâ facie* evidence of a lesion that takes the case out of the category of degenerative disease. In most acute cases there is other evidence of a focal lesion, extending, at some level, beyond the limits of the motor path. Such indications are initial impairment of sensation, or a girdle-pain. The latter indicates irritation of the posterior root-fibres at a certain level, and proves that at that level the disease extends beyond the limits of the pyramidal tracts. Spastic paraplegia is common after such lesions, but is secondary and not primary.

If any marked sensory symptoms exist in a case that presents the symptoms and course of primary lateral sclerosis, these are evidence that the sclerosis extends beyond the motor tracts, and the case is not one of pure lateral sclerosis. It is doubtful, however, what significance is to be attached to very slight sensory symptoms, such as slight subjective sensations of dull pain, formication, &c., when they exist alone. It is possible that they are due to functional disturbance in the sensory nerve-elements and do not imply structural disease outside the motor area. They usually remain stationary, and are not followed by any more pronounced sensory symptoms. Considerable muscular atrophy in any part is commonly regarded as bringing the case under the designation of "amyotrophic lateral sclerosis," but some cases of the kind, in which the wasting is confined to a few muscles, resemble more closely the cases we are now considering than they do those with widespread and extreme muscular atrophy. These cases are further considered in the account of progressive muscular atrophy.

The disease is not uncommon in those who are at the age and of the sex at which hysteria prevails, and there is no form of cord disease that is so often mistaken for hysterical paraplegia. The mistake is facilitated by the perfect muscular nutrition in most cases of spastic paraplegia. When there is considerable spasm, the mistake ought not to occur, as the extensor character of the spasm, described above, is distinctive; nothing resembling it ever occurs in hysterical paraplegia. Hysterical contracture is fixed, and does not vary with posture as does the "clasp-knife rigidity" of spastic paraplegia. When the spasm is trifling or absent, as in slight and early cases, the diagnostic difficulty is much greater, and is increased by the fact that slight excess of myotatic irritability occurs in some cases of so-called hysterical paralysis. But this scarcely ever reaches the degree necessary to give rise to a true foot-clonus or a rectus-clonus. There may be a "spurious foot-clonus," or a true clonus may be obtainable if there is hysterical contracture, but apart from such contracture a true foot-clonus or a rectus-clonus deserves the greatest weight, as all but conclusive evidence of organic disease. I have known many mistakes in diagnosis, in which lateral sclerosis was mistaken for hysterical paraplegia owing to disregard of the evidence afforded by this symptom, but I do not recall a single instance in which the opposite error resulted from undue regard to this symptom. When there is an excess of myotatic irritability in so-called hysterical paralysis it is a persistent symptom and must depend on more than functional disease. There must be changes in nutrition, and consequent persistent defective control of the muscle-reflex centres. On the hypothesis that I have advanced, this control is exerted by the termination of the pyramidal fibres, *i. e.* of the upper motor segment, by the structures, degeneration of which may probably cause spastic paraplegia. A case is actually on record, the case of lateral sclerosis observed by Charcot in 1865, in which an initial

hysterical paraplegia, cured suddenly, and relapsing on emotion, passed into lateral sclerosis.

When the arm and leg suffer on one side only, the disease may be mistaken for cerebral hemiplegia. The resemblance may be very close. There is not, however, any affection of the face, which, although theoretically conceivable, is, as a matter of fact, always absent. The limbs on the other side are never normal, but present slight symptoms. The excess of myotatic irritability in the other arm has afforded, in all the cases I have seen, a distinct indication of the nature of the malady. It is never present in simple cerebral hemiplegia, although such excess is often present in the leg of the unparalysed side.

The diagnosis of the congenital infantile form is only difficult when the observer is unaware of the occurrence of these cases. A slight degree of inco-ordination in the hands will usually be found if they are carefully watched while the patient takes hold of some object. The wide separation and irregular movement of the fingers is very characteristic. Chronic primary cord diseases are almost unknown in young children. Caries of the spine, causing paraplegia, is, in them, always recognisable, and the distinct onset of the paralysis, in a previously healthy child, is an absolute distinction from the cases of birth-palsy. The same fact also distinguishes glioma of the pons, which may likewise cause spastic paralysis of arms and legs.

The disease with which the infantile form is most frequently confounded is pseudo-hypertrophic paralysis, to which a superficial resemblance may be caused by the gait of slight cases, the large muscles, and the contracture of the calves. Besides the other characteristic features of the muscular malady, the two are distinguished especially by the excessive knee-jerk of spastic paraplegia, by the active and yielding nature of the contracture, and by the course of the disease; the impairment of locomotion gradually lessens in birth-palsy while it increases in pseudo-hypertrophic paralysis.

PROGNOSIS.—In primary lateral sclerosis the chance of recovery and the danger to life are both small. There is some prospect of arrest, and even of slight improvement, if the disease has not reached an advanced stage. When the spastic state is well developed and has lasted for some time it is very rare for marked improvement to occur. This is perhaps due rather to the tendency of the disease than to its degree, since a similar condition that is secondary to a focal lesion may undergo remarkable improvement. In slight cases there may be considerable gain of strength. In some cases which improve it is possible that the disease is limited to the terminal structure. In the infantile form, the prognosis is much better. Most children ultimately gain some power of walking, and in slight cases the ultimate impairment may be trifling.

TREATMENT.—So far as drugs are concerned, the treatment is the same as that of posterior sclerosis already described. The drugs most

useful are the same, but, unfortunately, their influence is less frequently appreciable. *Nux vomica* and strychnine have, however, to be given with caution, and in very minute doses, as they have a tendency to increase the spasm. In severe cases this is a most distressing symptom, and often not amenable to any influence. Bromide sometimes lessens it slightly, but even large doses of bromide have but a trifling effect. Neither Indian hemp, belladonna, calabar bean, or *piscidia erythrina* has much effect. Absolute rest is sometimes of service, and occasionally seems to produce improvement, which all treatment failed to effect while the patient was walking about. Rubbing is also beneficial in some cases. Its influence on the spasm is often very distinct during the process, and a long course of rubbing has produced a permanent improvement in the spastic condition. Upward rubbing seems to have more influence than kneading the muscles. If there is contracture of the calf-muscles, the foot should be pressed up while these muscles are rubbed. When there are facilities for it, the rubbing may advantageously be combined with sweating in the Turkish bath. In one case, of moderate degree, almost all the symptoms passed away after a long course of Turkish baths, arsenic being also given. The patient, who could at first walk scarcely half a mile, became able to walk several miles without fatigue. Electricity is useless in the pure disease. Faradism, and all painful applications, are harmful, stimulating further the already excessive excitability of the reflex centres. The constant current to the muscles, or from the spine to the muscles or to the feet in water, has no influence in lessening the spasm or improving the strength, as far as I have been able to observe, and I have tried it thoroughly in many cases.

In the infantile form, drugs are useless. Rubbing is desirable, and can be efficiently performed by the nurse or mother. Carefully planned gymnastic exercises are also useful. The tendo Achillis is sometimes divided for the contracture of the calf-muscles, but the operation is useless and ought never to be performed. Supports occasionally help the child to walk somewhat sooner than it would without their aid, and so may hasten improvement, but they should only be employed when there is sufficient power and control to enable them to be useful.

ATAXIC PARAPLEGIA

(COMBINED LATERAL AND POSTERIOR SCLEROSIS).

The term ataxic paraplegia seems the most accurate clinical designation for a disease of the spinal cord which presents a combination of the symptoms of paraplegia and ataxy, and consists in combined disease of the posterior and lateral columns. Although its clinical features present some varieties, and may approach those of each of its constituent forms of disease, yet in the majority of cases the symptoms are uniform, and sufficiently characteristic to justify the distinction of the disease and its separate description. A few pathological observations have been published.* The following account is based on these and on a series of cases that have come under my own observation, and of which the clinical characters were well marked.

CAUSES.—Neurotic heredity is to be traced only in a small proportion of the cases, about one tenth. A history of syphilis is as rare as it is frequent in pure tabes. Males suffer much more frequently than females. The disease usually commences between thirty and forty, but I have known it to begin as early as nineteen and as late as fifty-two. Exposure to cold is to be traced occasionally as an exciting cause. In one young lady, the symptoms commenced after a season of balls, at which when heated she would habitually sit at open windows, and often sleep in clothes saturated with perspiration. A severe concussion of the spine has sometimes preceded the first symptoms by a few months. The disease may also follow great sexual excess. In many cases no cause can be traced, immediate or remote. Its general etiology thus resembles that of simple spastic paraplegia.

SYMPTOMS.—The onset of the disease is usually slow and gradual, two or three years passing before walking power is much impaired; it is rarely subacute, so as to reach a considerable degree in two or three months. The legs are the first to suffer, and the symptoms may remain limited to them or may involve the arms also. There is a slow and simultaneous development of weakness and defect of co-ordination. The patient finds that he tires more readily than before, and also becomes unsteady on turning or walking in the dark, and these symptoms gradually increase. Examination then shows distinct loss of power in the legs. This may be slight and difficult to elicit, but if all the movements are tested it will be generally found that there is marked weakness of the flexors of the knee and hip, often much greater than that of the extensors. Frequently one leg is distinctly weaker than

* By Prevost, 'Arch. de Physiologie,' T. iv; Pierret, *ib.*; Babesien, 'Virchow's Archiv,' Bd. 76; Kahler and Pick, 'Arch. f. Psychiatrie,' Bd. viii; and Westphal, *ib.*, Bd. viii and x. Westphal's observations are the most important.

the other. The patient is unsteady when he stands with feet together, and he tends to fall if the eyes are then closed. If the feet are bare, the irregular action of the muscles is shown by the movement of the tendons on the dorsum of the foot, as in tabes. In most cases the inco-ordination is revealed also by the patient's gait, which is distinctly unsteady. There is rarely the high movement and sudden descent of the feet often seen in tabes, although I have observed it in a few cases. But the walk may closely resemble that of many cases of tabes; the patient is unsteady, reels on turning, and has often to bring his foot suddenly to the ground to maintain his equilibrium. He may even have to steady himself with a stick, or to catch hold of some adjacent object, to save himself from falling. The ataxy is equally evident when he lies, and attempts, his eyes being closed, to touch some object with his foot.

The sensory and reflex symptoms present a marked contrast to those of tabes. Lightning pains are almost always absent; I have only met with them in one case, and in this they were a transient symptom. Sometimes there is a slight dull pain in the legs, felt especially on fatigue. Dull pain in the sacral region or in the spine is not uncommon, and is often an early symptom. The sacral pain, indeed, is sufficiently frequent to deserve special note. A girdle-pain is met with only in rare cases. As a rule there is no loss of sensation either on the legs or trunk. Hyperæsthesia is equally rare. Reflex action from the sole may be normal or increased; less commonly it is diminished. The cremasteric and abdominal reflexes are sometimes lost. The most striking difference from tabes, however, is in the condition of myotatic irritability, which, in the vast majority of cases, is greatly increased. The knee-jerk is quick and extensive; it can be obtained from above (see p. 12), and there is generally a distinct rectus-clonus. The foot-clonus is also commonly to be obtained. This myotatic excess usually persists throughout the whole course of the disease, whereas, in ordinary tabes, however slight the lesion, the knee-jerk is all but invariably lost.

The arms may be normal, or may present symptoms similar to those in the legs, inco-ordination, weakness, and marked excess of the myotatic irritability. The muscles in both arms and legs are usually well nourished.

Sexual power is often lost early in the disease. The sphincters are frequently impaired, sometimes early, but the impairment rarely reaches a considerable degree.

The iris usually acts to light, but I have seen loss of the light-reflex in two or three cases and have once known accommodation to be lost, the action to light being normal. Optic nerve atrophy occurs only in rare cases, less frequently than in tabes; I have seen one case in which there were indications of a retro-ocular axial neuritis. The external ocular muscles are usually normal, and there is no nystagmus.

Slight impairment of articulation is not uncommon; sometimes there

are irregular tremulous movements of the face resembling those of general paralysis, and this in cases in which there is no mental change. Westphal has observed marked ataxy of the facial muscles. As a rule the mental state is either normal or there is merely slight failure of memory. Undue frequency of the pulse is occasionally present, but is less common than in tabes.

As the disease increases the muscular power becomes more and more impaired, usually without much increase in the inco-ordination, which sinks into the background as the paralysis increases, and the patient has to have recourse to supports which at the same time afford him guidance. The increased myotatic irritability continues; stiffness and rigidity develop as its consequence. Thus the aspect of the patient comes to be that of spastic paraplegia; the feet drag as the patient walks, shake from clonus when he stops, and the legs are hauled forwards with visible effort at each step. Indications of unsteadiness may still be observed in isolated movements. The arms, if previously free, may begin to suffer, but sometimes they escape altogether. The motor weakness may go on to complete paralysis. This is quickly reached in some cases, but so slow is the usual progress of the disease that one patient in whom the disease has at no time been absolutely stationary, is still able to stand, whose case I have had under observation for eight years. In spite of the progress of the weakness, sensation remains in most cases unimpaired, and the reflex action of the iris, unless early lost, continues perfect throughout.

The symptoms present, in some cases, variations from this type. Sensation on the legs may be impaired and the knee-jerk may be lost. These cases are intermediate between the form now under consideration and pure tabes. They are, however, very rare, and do not constitute more than five per cent. of the cases in which ataxy and weakness come on together.

The disease has little tendency to cause death. Indeed, the fatal cases have, for the most part, been untypical, and do not convey an accurate idea of the characters of the disease. The chief danger to life is from the accidents common to all chronic spinal affections, —kidney disease and bedsores.

Among complications the most important are mental changes resembling those of general paralysis of the insane, of which indeed this combined sclerosis may form part. Slight muscular atrophy sometimes occurs. Arthritis of doubtful significance has been once observed. A patient of Westphal's presented derangement of the sympathetic, and died from peculiar spasm of the muscles of respiration. Visceral crises, however, are practically unknown.

PATHOLOGICAL ANATOMY.—In all cases the spinal cord has presented sclerosis of both posterior and lateral columns; but the precise extent and degree of the degeneration are subject to considerable variations. As a general rule, the sclerosis of the posterior columns differs from

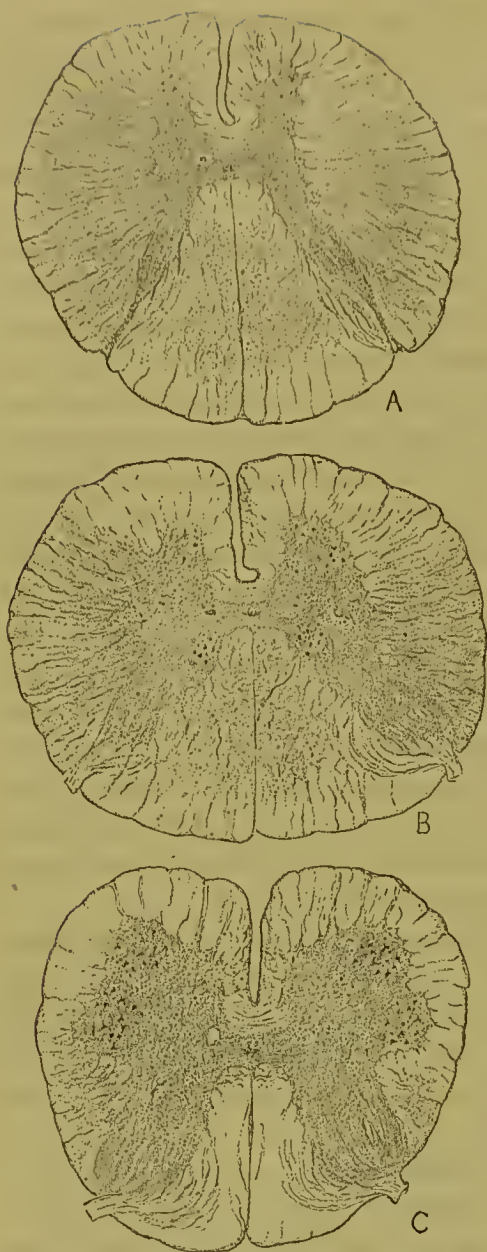


FIG. 121.—Ataxic paraplegia, combined lateral and posterior sclerosis. A, upper dorsal; B, last dorsal; C, mid-lumbar. The posterior columns are free from sclerosis in C, except in their anterior parts; in B the disease involves the middle and anterior parts of both post.-med. and post.-ext. columns except in the neighbourhood of the neck of the horn; in A the sclerosis is slighter and is confined to the middle three fifths of these columns. The sclerosis of the lateral columns in C is limited, on the right, to the pyramidal tract, on the left it extends in front of this; in B it is very dense in the whole lateral column, involving not only the pyramidal tract but the limiting layer, and part of the "mixed zone;" in A it is similar in extent but slighter in degree. There is some increase of tissue throughout the anterior columns, and a focus of sclerosis near the anterior fissure, on the right in A and B, on both sides in C.*

that of tabes in two particulars. First, it is not more intense, and often it is less intense, in the lumbar than in the dorsal region of the cord. Sometimes, indeed, as in the case shown in Fig. 121, in the middle and lower parts of the lumbar region, the posterior columns may be free from sclerosis, although it is considerable in the dorsal region and at the junction of this with the lumbar enlargement (B). The second difference is that the sclerosis has rarely that special intensity in the root-zone of the postero-external column which characterises the lesion of tabes. In rare cases the whole posterior column in the lumbar region is diseased. The part of the external column near the commissure and near the neck of the posterior horn usually remains free. Sometimes the degeneration does not extend up to the posterior surface of the cord, as in the case figured, in which it affects chiefly the middle three fifths of the posterior columns. When the degeneration is considerable, the posterior median columns, in the upper part of the cord, may present the usual ascending degeneration of secondary origin. When the lesion is slight in degree in the lower

* I am indebted to Mr. J. Hopkins for the opportunity of drawing these sections. The patient was a man aged twenty-one, in whom weakness of the legs commenced at twenty, after a wetting, improved, and then slowly increased. He was admitted

half of the cord, there may be only a slight diffuse degeneration of the columns in the cervical region, similar to that elsewhere, and not the more intense limited affection of the median part, which occurs when there is a considerable lesion of the column lower down, and characterises the secondary form.

The degeneration in the lateral columns is also variable in extent and position, and is not often "systemic" in character, *i. e.* does not correspond to a single system of fibres. In one or two cases, indeed, the whole pyramidal tract, and this alone, has been degenerated, but more often the sclerosis, while intense in the pyramidal tract, also extends in front of this into the mixed zone of the lateral columns (Fig. 121, A, B). (A similar extension, it may be noted, is met with in the sclerosis that attends degeneration of the anterior cornua; compare Fig. 119.) The lateral limiting layer, between the pyramidal tract and the grey matter, may be also invaded. The direct cerebellar tract often escapes, as in Fig. 121, A (B is below its level of origin). But in some cases there is a zone of sclerosis round the periphery of the cord, extending deeply into the lateral column in the position of the pyramidal tract. The latter has been found diseased, in some degree, in all cases, and in several the extent of its degeneration has been greater in the lower part of the cord than in the upper. As in most other degenerative diseases of the cord, there may be some increase of connective tissue in the unaffected columns, and it may be so intense at some spots as to constitute areas of distinct sclerosis, such as are represented near the anterior median fissure in Fig. 121. As a rule no morbid change has been recognised in the grey matter or in the membranes. In one case only was there slight meningitis. Disease of the ascending root of the fifth was found in a case in which there were corresponding symptoms. The peripheral nerves seem not to have been examined in any case. The muscles have been found normal.

PATHOLOGY.—The double lesion in the posterior and lateral columns supplies an explanation of the two sets of symptoms which characterise the disease. The inco-ordination is doubtless due chiefly to the disease of the posterior columns of the cord. There is an obvious analogy between the symptoms and lesion in this disease and in true tabes. But there are also differences between the two diseases in this particular. We have seen that in tabes the ataxy is probably due chiefly to the disease of the sensory muscle-nerves. In ataxie

a year after the onset, with considerable weakness of the legs, a reeling, unsteady gait, increased knee-jerk, foot-clonus, but with no anæsthesia, wasting of muscles, pains, or affection of the arms. The symptoms slowly increased, paraplegia became absolute, and the spasm very intense, sometimes flexor and sometimes extensor. There was a doubtful impairment of sensibility to touch on the legs, but no loss to pain. The sphincters became affected, bedsores formed, and the patient died two years after the onset. Fuller details will be found in 'Brain,' October, 1883, p. 383.

paraplegia, to judge by the general integrity of sensation in the legs, and by the preservation of myotatic irritability, the sensory nerves escape and among them those of the muscles. In correspondence with this integrity of sensation, the root-zone in the lumbar region scarcely ever presents dense sclerosis, and is often free from degeneration. Hence one element in the production of inco-ordination in tabes is absent in ataxic paraplegia. But we have seen that, in the former disease, other mechanisms are influential in causing this symptom, and these are doubtless effective in the malady we are now considering. The considerable disease of the posterior columns in the dorsal region, and the occasional degeneration of the direct cerebellar tract, may arrest conduction upwards from the muscles to the cerebellum. As we have seen, it is probable that the direct cerebellar tract conducts impressions from the lower trunk-muscles, and that it is also probable that most of the fibres of the posterior median columns have a similar function, conducting from the muscles of the legs impressions which reach the cerebellum through the connection with it of the post-pyramidal nucleus of the medulla. If the impressions thus conducted guide cerebellar co-ordination, we can understand that ataxy may result from the interruption of this path. That interruption of some conducting path in the cord will produce inco-ordination is proved by the occurrence of this symptom in cases of unilateral lesion of the cord, on the side of the lesion (see p. 157, note). Thus the chief difference between the two diseases, so far as concerns the effect of the lesion which causes the inco-ordination, is that in tabes it arrests the muscle-reflex action. The sclerosis in ataxic paraplegia occupies also the region of the cord in which the short vertical fibres run, connecting the posterior grey matter at different levels, and it is possible that the damage to these fibres may contribute to the ataxy.

The only recognisable lesion that can be regarded as the cause of the paralysis is the degeneration of the pyramidal tract. This is, indeed, so variable in degree in different cases, and in some is so small in proportion to the degree of palsy, that it has been questioned whether this lateral degeneration is the sole cause of the paralysis. But no other lesion has been found; the excess of myotatic irritability and ultimate spasm are conclusive evidence that the cause of the palsy is in the upper segment of the motor path, *i. e.* above the ganglion cells of the anterior cornu (see pp. 116 and 144), and when the paralysis has been greater in one leg than the other, the disease of the lateral column has been found to be greater on the corresponding side. It is possible, however, that the degeneration is often more extensive in the termination of the upper segment in the grey matter, than in the (pyramidal) fibres of this segment, and that the occasional discrepancy between the amount of degeneration of the lateral column and the amount of paralysis may be susceptible of the same explanation as in pure spastic paraplegia (see p. 329). In most cases that have been examined, the visible disease was greater in the lower than in the upper

part of the cord, *i. e.* greater in the lower than in the upper part of the segment, increasing from above downwards. This suggests that the maximum lesion is at the *lowest* part—in the terminal ramification within the grey matter. Degeneration here would not necessarily cause any secondary change in the white columns, *i. e.* in the part of the segment above its termination, while it would explain all the paralytic and spastic symptoms of the disease.

The differences between this disease and tabes depend therefore on the escape of the sensory nerves, both in their peripheral course and in the posterior column of the cord. It is on the integrity of the reflex arc that the preservation of myotatic irritability depends, not on the presence of a lesion in the cord tending to increase this reflex action. If the knee-jerk is lost as in tabes, it is not restored by degeneration of the pyramidal tracts, whether beginning in the cord or descending from the brain. Hence this difference from tabes depends partly on the escape of the peripheral nerves, partly on the difference in the distribution of the sclerosis in the posterior columns, by which the root-fibres are damaged in one case and escape in the other.* It is not surprising, however, that intermediate cases should be met with, in which these differences are not present; in which the sclerosis extends to the root-zone, and lightning pains, and some loss of sensation, show that the fibres of the posterior roots from the skin are involved in some degree. Even in these cases, however, the knee-jerk is not lost; if it is absent there is no spasm and the cases are then of a different character; they are cases of tabes with secondary lateral sclerosis. It is in the remarkable facts that, as a general rule, if the knee-jerk is not lost early it is not lost at all, and that if it is lost early, spasm does not come on, that we have the justification for the separation of these cases from tabes.†

It has been doubted whether the disease is to be regarded as a system degeneration, in the strict sense of the word. The lesion of the lateral columns certainly, as Westphal has insisted, does not correspond strictly to the pyramidal tracts, and yet its vertical extent forbids us regarding it as focal in nature. The disease is certainly allied to system-diseases, and its claim to be considered such may perhaps be strengthened by future observations as to the elements which compose the more extensive degeneration,‡ but its claim to

* We have seen (p. 134) that the fibres that subserve reflex action cannot be the same as those that ascend in the posterior median column, and which, in this disease, are often damaged higher up the cord.

† If cases occur in which an ultimate extension of the sclerosis of the posterior column abolishes the knee-jerk and the spasm, it is quite certain that such cases are too rare to invalidate the grounds of distinction. The possible combinations of morbid processes are only limited by arithmetic and incompatibility, but this does not stultify our distinctions.

‡ For instance, it may be found that the degeneration in front of the pyramidal tract is of an association-system of short vertical fibres and that the annular sclerosis is of the direct cerebellar and antero-lateral ascending tracts.

separate distinction is at present, as we have seen, clinical rather than pathological, is based on the common uniformity of the range of symptoms, alike in different cases, and in the same case throughout its course.

DIAGNOSIS.—The diseases from which ataxic paraplegia has to be distinguished differ according to the stage of the disease at which the diagnostic problem presents itself. The dominant symptom in the early period is the inco-ordination; in the later period, the spastic palsy. Hence the disease in the early stage is liable to be confounded with pure locomotor ataxy. In discussing the relation of the two diseases we have already considered their differences. Of these the most important is the condition of the knee-jerk, which is lost in the one, excessive in the other. In the rare cases of early tabes in which it is not lost, it is never increased. The presence of a clonus is also exclusive of pure tabes; and so also is the extensor spasm, of which we may often observe some indications, while power is still only slightly impaired. Other differences may confirm the diagnosis, but are never necessary to make it, and are indeed not uniform enough to be alone conclusive. In the state of sensation on the limbs, in the occurrence of, or freedom from, pains, and in the action of the iris, the two diseases present a contrast in the vast majority of cases, although not invariably. From primary spastic paraplegia the diagnosis depends on the presence of inco-ordination, or in a history of it if voluntary power has become too slight to be susceptible of derangement. + Ataxic paraplegia is spastic paraplegia, plus inco-ordination. If the ataxy is considerable, as it often is, the distinction is easy enough, for experience shows that it is chiefly with tabes that such cases are confounded. The so-called "hereditary ataxy" presents a close resemblance to ataxic paraplegia, and is indeed, as we shall see, intermediate between this disease and true tabes. It is distinguished by its occurrence in several members of the same family, by the common loss of the knee-jerk, and by the presence of nystagmus and of some impairment of articulation.

The greatest diagnostic difficulty is presented by cases which have a subacute onset, and the distinction from a local myelitis involving posterior and lateral columns may be very difficult. It depends chiefly on the progressive tendency of the symptoms, which contrast with the regressive tendency of myelitis. It is possible that when there is a degenerative tendency, a focal myelitis may set up a progressive degeneration in the columns concerned, as it certainly may set up a degeneration limited to the posterior columns.

A tumour in the middle lobe of the cerebellum may cause unsteadiness, closely resembling that of some cases of ataxic paraplegia, and it may also cause weakness of the legs with increased knee-jerk, from the pressure on the pyramidal fibres as they pass through the pons. In such cases indeed we probably have the two elements of the disease,

produced although in a different manner by a lesion of different position. We have a degeneration of the lateral columns which is secondary instead of primary, and we have disease of the co-ordinating centre instead of interruption of the path to it. But the weakness in the legs is never great in cerebellar tumour, and special symptoms of this are never absent. Occipital headache, vomiting, and optic neuritis are present in most cases. In a case in which the pons is compressed, some cranial nerves are usually also damaged.

PROGNOSIS.—So far as the prospect of recovery is concerned, the prognosis in ataxic paraplegia is unfavorable. In a few cases I have seen considerable improvement; and arrest is occasionally obtained, but in the majority of cases the progressive tendency of the disease baffles all attempts to check it. The downward progress of most cases is very slow, far slower than might be inferred from the history of recorded fatal cases, and the danger to life is small.

TREATMENT.—The treatment of ataxic paraplegia is the same as that of the allied diseases, and this need not be here repeated. The remedies most useful in tabes also most frequently do good in this disease. In the later period, in which spasm has attained considerable intensity, the treatment necessary is that of the condition described in the preceding chapter. I have known a long course of Turkish baths, with thorough rubbing, in more than one case to be of marked service, and distinctly contribute to the improvement that occurred. There are no symptoms peculiar to ataxic paraplegia that require special treatment.

“ HEREDITARY ATAXY ”

(FRIEDREICH'S DISEASE, HEREDITARY ATAXIC PARAPLEGIA).

The so-called hereditary ataxy is a form of ataxy, or rather of ataxic paraplegia, usually depending on combined posterior and lateral sclerosis, which occurs in families, and differs also from the common forms of tabes and of ataxic paraplegia, in the early age at which it always commences, and in the presence of certain additional symptoms. It is often termed “Friedreich's disease,” because this physician first described the characteristic features of the malady and the lesion in the posterior columns.* About sixty-five cases have been hitherto recorded.†

The dependence of the disease on a congenital tendency is clearly shown by its usual occurrence in families. But direct inheritance has

* Friedreich's first account was given to a medical society in 1861, and published in ‘Virchow's Archiv’ (Bd. 26 and 27) in 1863. Further cases were published by him in 1876 (ib., Bd. 68 and 70).

† The most complete list of these (57 cases) has been given by Dr. Everett Smith, Boston Med. and Surg. Journal, Oct. 15, 1885.

been traced in only a few instances. In one, the mother of the family affected, and *her* mother, both suffered from the disease. That a general neuropathic inheritance is also sometimes effective is shown by cases in which there is a history of other neurotic diseases, such as insanity, in collaterals or ancestors. Alcoholism in parents has been supposed to be influential, but the evidence of this is not strong. Consanguinity of parents has existed in some instances, and has doubtless intensified the morbid tendency in these cases.

The family tendency of the disease is shown by the affection, in most instances, of brothers and sisters. The sixty-five cases were distributed in nineteen families, giving an average of rather more than three to each. The number affected in one generation has varied from two to eight. In the case in which the mother and grandmother suffered, an uncle and seven children were also affected, making ten in one family. Scarcely any isolated cases have been recorded; whether this is because they do not occur, or because the nature of the disease has not been recognised, is uncertain. The two sexes present nearly equal liability; males slightly preponderate (thirty-five males to thirty females). In some families the two sexes have suffered equally, but in others the disease has shown a marked tendency to affect one sex. Thus in one family of nineteen, the two males suffered and the seventeen females escaped. In another instance, recorded by Musso, a brother and sister (the offspring of a melancholic mother) married healthy individuals; the brother had three daughters affected out of seven living children; and of the sister's children three sons were diseased. Another curious circumstance is that three of the brother's and four of the sister's children were born dead.

The age at which the first symptoms are recognised has varied between four and twenty-four years.* The seventh and eighth years of life are those in which disease most often begins, and next comes the period of puberty, from twelve to sixteen. It shows a slight tendency to begin earlier in males than in females, and often a marked tendency to commence about the same period in the same family. Immediate causes can rarely be traced.

SYMPTOMS.—The first and chief manifestation of the disease is a gradual impairment of co-ordination, first in the legs and afterwards in the arms. Initial pains scarcely ever occur. The ataxy is shown by unsteadiness in standing and walking, at first slight, but slowly increasing, until the feet have to be placed wide apart in standing, and the patient reels in walking like one under the influence of alcohol. The feet are not often raised too high, unless when an unusually long

* In the family recorded by Everett Smith, the father presented symptoms of ataxic paraplegia at the age of sixty-six, coming on gradually after an attack of rheumatism produced by exposure to cold. In the age of the sufferer, this case stands alone in the history of the disease, and is not included in the figures given. The case may be a mere coincidence, or may show that a latent predisposition may persist through life.

step is taken. Closure of the eyes causes a considerable increase in the unsteadiness in some cases; in others it has but little influence. The impairment of movement in the arms is of a similar character, but usually commences some time after that of the legs. There is irregularity in the voluntary movement of the arms and fingers, and the ataxy has often a distinctly jerky character. In most cases the power of the muscles is at first unimpaired, their nutrition is good, but the myotatic irritability is lost. In exceptional cases, as in one recorded by Selig-muller, it has been preserved and increased. In most cases the knee-jerk has been found absent as soon as the patient came under observation; in one case it disappeared after the other symptoms had set in.

As the disease progresses, some jerky irregularity develops in the movements of the neck and head, so that the head presents slight unsteady movements, sometimes like an irregular tremor, when it is supported by the muscles. Speech is also impaired; syllables are elided, and there may be, with this, an occasional hesitation. There is no twitching of lips, but occasional jerky movements have been noted in the tongue. The affection of speech is not often an early symptom. It may not be noticed until three, five, or ten years after the onset of the other symptoms. In most cases (but not in all) there is nystagmus when the eyes are moved laterally, due clearly to jerky unsteadiness in the action of the muscles, corresponding in the two eyes. Sometimes the movement is slower than in most forms of nystagmus, and it is rarely present when the eyes are at rest, directed straight forwards. This symptom also usually comes on after those in the limbs. Paralysis of the ocular muscles does not occur, nor does optic nerve atrophy.

Sensory symptoms are very variable. Lightning pains and any severe pains are extremely rare, but slight dull or rheumatoid pains in the legs are not uncommon. Sensibility has been quite normal in many cases; in others there has been slight anæsthesia in the legs, early in some instances, late in others. Sensibility to pain and temperature is very rarely impaired. Sensation may, however, be unaffected even when the ataxy and weakness have become extreme. The sense of posture of the limbs has been found normal in several cases in which it has been examined. The electro-sensibility of the muscles is said to be sometimes lessened. Increased sensitiveness to pain is occasionally met with. Reflex action from the sole is usually preserved, but may be lost when there is anæsthesia. The cremaster reflex is often lost. On account of the age of the patients little is known of the condition of sexual power, but it is certainly often lost. Menstruation becomes irregular and ceases. The sphincters, as a rule, are unaffected. There is no tendency to trophic changes in the skin or joints.

Although muscular power is commonly normal at first, and may be normal even when the ataxy is considerable, it usually becomes impaired as the disease progresses, and sometimes weakness comes on, with the

ataxy, at the onset of the disease. The loss of power is always greatest in the legs and may be confined to them. The flexors suffer more than the extensors. It may be ultimately great, although it rarely amounts to absolute paralysis. Slight wasting of the muscles may occur in the later stages of the disease, but is only attended with trifling depression of the electrical contractility. Lateral curvature of the spine, and talipes equinus, or equino-varus, of the foot, have been observed as later results of the muscular weakness, developing under the influence of posture, and contraction of the flexors of the knees has also resulted.

Visceral crises do not occur. Frequency of pulse has been noted, however, in many cases, and curious vaso-motor symptoms (flushing, œdema, sweating, polyuria, and salivation) were present in one of Friedreich's cases. There is no mental change that can be regarded as part of the disease. Imbecility co-existed in one case (Power).

The rate of progress of the malady varies much, even in different members of the same family. One in whom the disease commenced last may be unable to walk, while another who suffered sooner may be still in the early stage of the disease. Now and then the disease is stationary for many years. The duration of the malady is correspondingly variable. It is always long, even more than thirty years, and in many instances it has not apparently shortened life. On the other hand death may occur at the end of ten or twelve years. The end has generally come from intercurrent affections, rarely from exhaustion. An attack of acute myelitis ended life in one of the cases recorded by Everett Smith.

PATHOLOGICAL ANATOMY.—The lesion in hereditary ataxy is that of ataxic paraplegia and of tabes combined. There is degeneration in the lateral column, often also in the anterior column, such as occurs in ataxic paraplegia, but the sclerosis of the posterior column is more intense than in that disease, and it is also more extensive, especially in the lumbar region. It is similar to the degeneration of tabes, and agrees with this also in that the posterior nerve-roots are usually affected, whereas in ataxic paraplegia they almost invariably escape.

The distribution of the lesions shows a close correspondence in different cases, as will be seen by a comparison of the figures here given, which are from three different cases of the disease. The sclerosis of the posterior columns may be complete throughout the cord, with the exception of a narrow band near the neck of the cornu, which always remains but little damaged. In the lumbar region it is usually intense up to the head of the horn and the posterior surface of the cord. Occasionally, however, it is somewhat less intense in the lumbar region than it is higher up, extending, however, into the root-zone. In the cervical region the sclerosis may also be general, or it may be greatest in the root-zone and in the posterior median columns. The cervical root-zone never escapes, as it often does in tabes.

The degeneration of the lateral columns always involves the pyramidal tracts, and is most intense in their position. It is not, however, limited to them. It usually extends outwards to the periphery of the cord, where the pyramidal tract does not reach the surface, thus involving the direct cerebellar tract, and it also extends forwards at the surface, so as to constitute a band of annular sclerosis, which varies in thickness in different parts of the cord and in different cases. In the inner part of the anterior column there may be a distinct area of degeneration in the position of the anterior pyramidal tract. This is seen in Fig. 123, in which this tract extends unusually low in the cord, and is distinctly degenerated in the upper part of the lumbar enlargement. Atrophy of the posterior vesicular column has been found associated with that of the direct cerebellar tract. Slight abnormal appearances have been seen in the nerve-cells of the

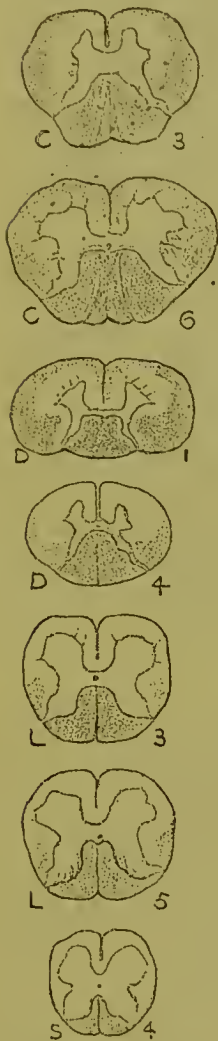


FIG. 122.—Hereditary ataxy; distribution of degeneration in the white columns, indicated by the dotted shading. (After Friedreich.)

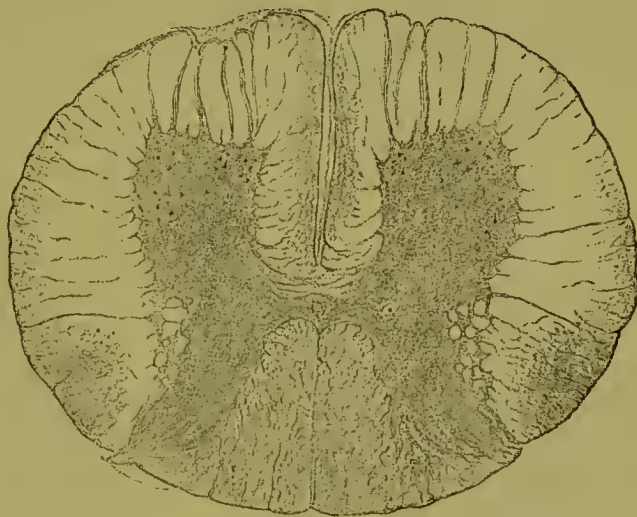


FIG. 123.—Hereditary ataxy; section of spinal cord at first lumbar segment. Sclerosis of the whole of the posterior columns except a narrow zone adjacent to the neck of the horn. Degeneration also of the lateral pyramidal tracts, and, in front of this, slight degeneration in the superficial layer of the lateral column. Adjacent to the anterior median fissure there is also a zone of sclerosis of the anterior pyramidal tract which extends, in this cord, into the lumbar region.*

* I am indebted for this section to Dr. Everett Smith, who has published the case in the ‘Boston Med. and Surg. Journal,’ Oct. 15, 1885. The patient was one of three sisters affected with the disease, the brothers being healthy. The father suffered from ataxic paraplegia late in life, as mentioned on p. 350. In the case from which the drawing is made, the inco-ordination began in the legs at the age of nineteen, and in the arms soon afterwards, and was quickly followed by loss of power, which gradually increased to almost universal paralysis, with muscular contractures (talipes equino-varus, &c.), and considerable loss of sensation. There was nystagmus, affection of articulation, and some mental dulness. Death occurred at the age of forty.

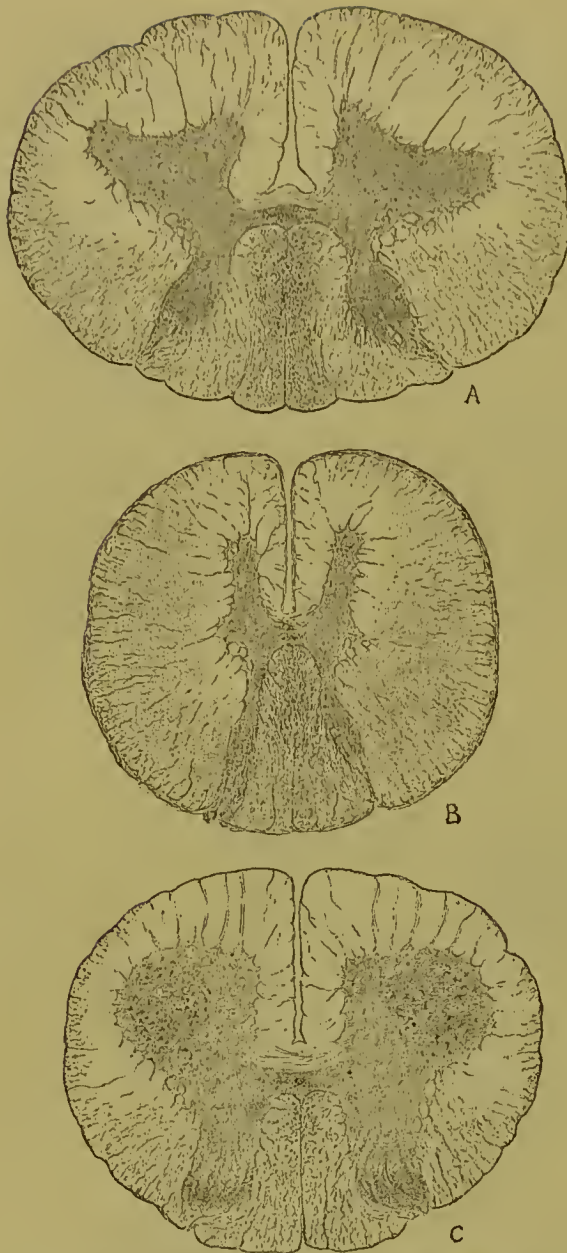


FIG. 124.—Hereditary ataxy. A, cervical; B, dorsal; C, lumbar regions of the cord. The posterior columns are sclerosed in their whole extent, except in the vicinity of the neck of the horn in the cervical and lumbar regions, the degeneration being rather less intense in the lumbar than in the other parts. In the antero-lateral columns there is an annular zone of sclerosis in the periphery of the cord, widening in the region of the pyramidal tract. In front of this tract the zone is widest in the dorsal region, and trifling in degree in the lumbar. The grey matter is but little affected.†

† For the sections from which the drawing is made I am indebted to Dr. Pitt (who prepared them) and Dr. Moxon, under whose care the patient died in Guy's Hospital. The case (full details of which will be found in the current volume of the 'Guy's Hosp. Reports') is one of a series of five cases (four brothers and a sister) which I reported some years ago to the Clinical Society ('Clin. Soc. Trans.,' vol. xiv, 1881, p. 1.)

anterior cornua, in some cases, but, as a rule, these are normal, and the grey matter presents no other disease. It is very likely, however, that future observations will reveal the occurrence of changes in the nerve-cells of the posterior horns, such as are met with in tabes.

The pia mater over the posterior columns has been found thickened. The posterior nerve-roots usually present some degeneration of their fibres; this may be partial (Fig. 125, B), or may amount to total destruction of the fibres (as at C). The peripheral nerves in the limbs have been found normal.*

Some general shrinking and induration of the pons and medulla were found by Friedreich, with atrophy of the cells of the post-pyramidal nucleus, and some degeneration of the restiform bodies, but none of the anterior pyramids. Corpora amylacea were present in the hypoglossal nerves.

PATHOLOGY.—The disease apparently occupies a clinical and pathological position between the combined sclerosis described as ataxic

* Friedreich, 'Virchow's Archiv,' Bd. 70, p. 145.

paraplegia and simple tabes, resembling the former in the common affection of the lateral columns and the consequent weakness, but differing from it and resembling tabes in the degree of affection of the posterior columns in the lumbar regions and the affection of the posterior roots; with these the loss of the knee-jerk and the affection of sensation are apparently connected. Isolated cases presenting the same combination of spinal symptoms are occasionally met with. The precise origin of the affection of articulation and of the nystagmus has not yet been traced.

From the age at which the disease commences, long before the period at which ordinary degenerations occur, we must look for its ultimate cause to a congenital tendency of development, by which the affected elements have a briefer period of vital endurance than the other tissues of the organism. Mobius has suggested that there is an actual arrest of development of these structures, but it is doubtful whether this assumption is warranted by the facts.

DIAGNOSIS.—In most cases the inco-ordination is sufficiently predominant to suggest that the disease is a form of ataxy, and the unsteadiness of the head, the affection of articulation, the nystagmus, and the age at onset, suffice for the distinction from ordinary tabes. The common form of ataxic paraplegia bears a close resemblance to this disease, a resemblance that is more than superficial, although the excessive knee-jerk and foot-clonus, almost always present in ataxic paraplegia, are usually absent in hereditary ataxy, and nystagmus is absent in the former. The difference in the state of myotatic irritability is, as we have seen, not absolute. Cases of combined lateral and posterior sclerosis occur, of tabetic type, in which the knee-jerk is lost, and in at least one case of hereditary ataxy the knee-jerk has been excessive. The age of onset and family multiplicity may decide



FIG. 125.—Hereditary ataxy; degeneration of posterior nerve-roots (from the same case as Fig. 124). A, normal anterior root, for comparison. B, posterior root, partially degenerated; a few normal nerve-fibres are seen, but most of the spaces which should contain nerve-fibres are empty. C, posterior root, totally degenerated, probably by a more acute process than that of B, since there is some destruction of the septa and increase of amorphous connective tissue. The products of degeneration are rendered invisible by the mode of preparation. (Compare C with the similar changes in a totally degenerated peripheral nerve shown in Fig. 51, B, p. 94.)

the question, but isolated cases occur, commencing soon after puberty, scarcely distinguishable from the hereditary disease. Such a case has been recorded by Kahler and Piek, which has been regarded by some, and not by others, as an example of this disease.

Disseminated sclerosis presents inco-ordination, nystagmus, and impaired articulation, but the ataxy of the arms differs in the wide range and violent character of the disordering jerks which characterise this disease; in the legs, moreover, simple unsteadiness is very rare. The affection of speech differs from that of hereditary ataxy in being simply "staccato," with undue separation of syllables, which are rarely run together. The cases are always isolated.

Cerebellar tumour and hereditary ataxy can hardly be confounded, in spite of the fact that the unsteadiness in walking is very similar in the two diseases; the common affection of the arms in the one, and the conspicuous head symptoms (severe pain, optic neuritis, &c.) of the other, sufficiently distinguish them.

PROGNOSIS.—The prognosis in every case is very serious, since the disease is essentially progressive, but life may be prolonged for many years, and in slight cases the malady may interfere comparatively little with the patient's occupation. Thus I have known a man, with very marked symptoms, follow his business as a tradesman for many years. The only guide to individual prognosis is the observed rate of progress.

TREATMENT.—As in other diseases that depend on a congenital tendency, treatment is almost powerless. The measures recommended for ordinary locomotor ataxy (apart from those suggested by the syphilitic relation of tabes) are those most suitable to the hereditary form. Arsenic and nitrate of silver especially deserve a trial; now and then they seem to check the progress of the disease for a longer or shorter time, but its progressive tendency ultimately reasserts itself.

CHRONIC SPINAL MUSCULAR ATROPHY

(PROGRESSIVE MUSCULAR ATROPHY; WASTING PALSY; AMYOTROPHIC LATERAL SCLEROSIS; CHRONIC POLIO-MYELITIS).

The disease which has long been known by the names "progressive muscular atrophy," and "wasting palsy" is characterised by slow wasting of the muscles, beginning in some one part, and usually spreading and increasing, until it is wide in extent and extreme in degree. The changes in the muscles depend on changes in the spinal cord, a slow degeneration of the ganglion cells of the anterior cornua,

accompanied by a similar degeneration in the motor nerve-fibres which spring from the cells. With this degeneration of the cells and peripheral fibres, there is usually also associated a degeneration of the pyramidal tracts in the cord.

Chronic muscular atrophy was separated from primary paralysis by Sir Charles Bell, and was afterwards described more fully by Aran and Cruveilhier. Bell and Cruveilhier regarded it as a spinal disease. Aran (who first called it "progressive muscular atrophy") thought that it was primarily a disease of the muscles, a view that was afterwards held by Duchenne, and subsequently also by Friedreich. This opinion was supplemented by a theory that the wasting was due to a disturbance of the sympathetic. Lockhart Clarke first discovered the disease of the grey substance of the spinal cord, and Charcot specially associated the atrophy with the wasting of the ganglion cells. When the constancy of the spinal lesion was demonstrated, it was thought that every form of chronic muscular atrophy was of spinal origin, but this view has proved erroneous, and it has been shown that there are forms of muscular atrophy which are purely local and idiopathic. The morbid process is not confined to the spinal cord. Atrophy of the nerve-cells from which the motor bulbar nerves arise often accompanies that of the spinal cells, and "progressive bulbar paralysis" is then associated with "progressive muscular atrophy." Moreover, the degeneration of the pyramidal fibres can often be traced through the brain.

Two varieties of the disease have been distinguished by Charcot, who has been followed in this by most subsequent writers. In the one variety the disease is manifested only by the wasting of the muscles; in the other there are, in other parts than those much atrophied, the indications of degeneration of the pyramidal tracts of the cord. In the latter cases, Charcot regarded the degeneration of the pyramidal tracts as the primary lesion, and the affection of the cornua as secondary. He therefore termed the cases of the first class "protopathic," and those of the second class "deuteropathic," and gave to the latter the name "amyotrophic lateral sclerosis." In Germany, however, and especially by Leyden, doubts have been expressed as to the validity of the distinction between the two classes of cases, and as to the sequence of the lesions in the second class. These doubts are not without foundation. The subject will be discussed when the pathology of the disease is considered, and reasons will then be given for the course here adopted of considering the two varieties together, as essentially one disease.

CAUSES.—The disease is more frequent in males than in females, the proportion being about three to one. It is chiefly a disease of adult life, commencing usually between twenty-five and forty-five. I have known it to begin at fourteen and as late as seventy, but most cases of muscular atrophy in early life are idiopathic, and not spinal.

Heredity is to be traced only in less than half the cases, and generally as an indirect neuropathic disposition. Rarely there is direct inheritance of the disease. Among instances that I have met with are a lady whose mother died from a similar atrophy, and another case (from which Fig. 135 is taken) in which a brother had died from some chronic disease of the cord, attended with wasting. When many members of a family suffer from muscular atrophy, the malady is nearly always idiopathic and not spinal. The affection occurs in all classes of society, and it is doubtful whether workers with the muscles furnish a larger proportion of the cases than can be accounted for by their greater exposure to the certain exciting causes.

Of these more direct causes one of the most frequent is mental distress and anxiety, and this is especially met with in females. A severe fright has been thought to produce it. Another cause is exposure to wet cold, which is a cause also of many other chronic spinal diseases. Sometimes the exposure has been habitual; sometimes a single exposure has been the cause, and some neuralgic pains, indicative of the deleterious influence on the nervous system, have followed the exposure and connected it with the later wasting. The pain has been sometimes in the part afterwards wasted, sometimes in some other more common seat of neuralgia. Thus one patient, after remaining in wet clothes for twelve hours, suffered for six weeks from severe neuralgia in the left side of the face, and then the muscles of the left shoulder began to waste. Although excessive use of individual muscles may cause them to waste, it is doubtful whether this influence produces general muscular atrophy. Injury to the cord, such as results from concussion of the spine, is a rare cause. It more often causes disseminated myelitis, which may be manifested by muscular wasting combined with other symptoms. In a few instances I have known progressive atrophy, of typical characters, to slowly follow a concussion, as if this had set up a perversion of the nutrition of the nerve-elements. Still more rarely a fall, injuring one limb, has been followed by muscular atrophy commencing in this limb and becoming general. Thus a woman fell downstairs, and pitched on her left hand and wrist; she had pain in the arm for a long time, and two years after the fall this arm began to waste and the atrophy ultimately became general. The relation might be passed as an accidental coincidence, were it not that in other central diseases, such, for instance, as paralysis agitans, the symptoms may commence in an injured limb.

The disease sometimes succeeds syphilis, and no other cause may be traceable. As with other degenerative diseases, an interval of years elapses between the primary disease and its nervous sequel. The cases that I have seen after syphilis have been typical in course and evidently degenerative in nature. It is probable that syphilis has some share in the causation of these cases, because the same relation obtains in the case of other diseases of the same class, and especially in that of locomotor ataxy. Occasionally, syphilis and a neurotic

heredity can both be traced. In rare cases the disease has followed acute specific diseases, especially measles, as in a remarkable group of cases, in one family, recorded by Ormerod.* General muscular atrophy may result from lead-poisoning, but this form is not, as a rule, progressive in character when its cause has ceased to act. In many cases of progressive muscular atrophy no cause for the disease can be traced.

SYMPTOMS.—The definite symptoms of the disease are often preceded by aching pain in the part afterwards wasted, rarely severe. Sometimes there is such pain in the spine or elsewhere, especially, as already stated, in the cases that are due to exposure to cold. Rarely some sensation other than pain precedes the local symptoms. Thus in one case a sense of coldness preceded the atrophy in each limb that was attacked. Weakness and wasting usually come on together; sometimes one and sometimes the other first attracts the attention of the patient. In the upper part of the arm the loss of power is usually first noticed, and in such covered parts the wasting may become considerable before it is discovered. In the hand the wasting is often first noticed, but sometimes it is the impairment of some delicate action, such as writing, that draws attention to the part. The affected muscles lose their proper shape, and there is flattening, or even a depression where there should be a prominence. If a patient is fat, however, the wasting may cause at first very slight alteration in external aspect.

The disease commences in the arms in nine tenths of the cases, and as frequently in one arm as in the other. It begins with almost equal frequency in the hand and in the shoulder-muscles. From the part first affected the disease spreads to other parts of the same limb. Before it has attained a considerable degree in one limb it usually shows itself in the corresponding limb on the other side; often in the muscles corresponding to those in which it commenced, sometimes in those affected second in order of time. As the muscles waste, the voluntary power is lessened, and paralysis results of various character and degree, corresponding to the atrophy. In the hand, the thenar muscles and interossei are usually the first to suffer, sometimes one, sometimes the other, but both are soon involved in most cases. The thenar eminence becomes flattened; the base of the first metacarpal bone becomes distinct and even prominent. Of the interossei, the atrophy of the abductor indicis is especially conspicuous; the normal prominence gives place to a hollow beside the metacarpal bone when the thumb is abducted. Depressions form between the metacarpal bones on the back of the hand, and also between the flexor tendons in the palm in consequence of the wasting of the lumbricales. The forearm-muscles may be next involved, the flexors usually before the extensors, and with the flexors the supinators may suffer or they may escape until the biceps is involved. Occasionally the disease begins in

* 'Brain,' 1884, p. 334.

the forearm, and then especially in the extensor muscles. The several parts of the long flexors or extensors may suffer unequally. Of the extensors, those towards the ulnar side usually suffer most. The exten-



FIG. 126. — Progressive muscular atrophy. Wasting of the muscles of the back and arms; in the forearm scarcely any muscular tissue can be recognised, and in the hand all the muscular prominences have vanished. (Drawn by Dr. H. R. Spencer.)

sors of the phalanges of the thumb are usually more affected than that of its metacarpal bone. Of the shoulder-muscles, the deltoid is generally the first to manifest the disease, and in some cases which begin in the hand the deltoid suffers before the forearm-muscles. The rounded contour of the shoulder becomes changed, and the head of the humerus can be recognised beneath the acromion. The wasting of the deltoid is soon followed by that of the other muscles of the upper arm and of the scapula. The triceps usually suffers less and later than the biceps, but sometimes the reverse is the case. The supra- and infra-spinati are often affected with the deltoid.

In most cases the wasting early involves the muscles of the back, and it sometimes begins in them. The middle and lower parts of the trapezius usually suffer first; the rhomboids and erectors of the spine at a later date. The affection of the trapezius is readily recognised if the patient tries to put the shoulder back. The highest part of the trapezius presents a remarkable indisposition to atrophy; it often remains intact to the last and then may contrast with the wasting below it, standing out, on each side, like a cord passing from the occiput to the shoulder. Hence Duchenne termed this part the *ultimum moriens*. I have, however, seen two otherwise typical cases (one beginning in the deltoids, the other in the hands) in which the highest parts of the trapezei suffered before the middle parts. The levator anguli scapulæ also generally escapes even when all the muscles about it are wasted. The serratus, latissimus, and

pectoralis major are usually affected later. They may escape wholly or in part; isolated bundles of the pectoralis may waste, the intervening parts escaping, or either the upper or lower part of the muscle may atrophy alone. According to the affection of the muscles that are attached to the scapula, the position of the bone changes, and it becomes rotated under the influence of the muscles that are unaffected and unopposed. The muscles that extend the head on the spine often suffer in considerable degree, and from this

there results a difficulty in the carriage of the head (Fig. 128). It is habitually inclined a little backwards, so as to balance it on the spine with but little muscular exertion; if moved forwards, or if the occiput is touched, it falls so that the chin touches the chest. It can be brought back into its former position only with difficulty; the patient has to incline the trunk backwards, so as to bring the head nearly into the vertical position, and then, with a sudden contraction of the sterno-mastoids, and a jerk, the head goes back into its former posture. The increased innervation of the weak extensors of the head often causes a synergic over-action of the frontalis muscles, which are normally associated with the extensors (so that the eyebrows are raised when the head is put back to look upwards). In such cases the wasting of the muscles at the back of the neck causes the skin to lie in transverse folds when the neck is extended. The patient may be unable, when lying in bed, to move the head from side to side. The sterno-mastoids also are often wasted; either the sternal or clavicular part may be most affected. In striking contrast to the general wasting of the neck is the condition of the platysma myoides, which always escapes, and may become hypertrophied in a vain attempt at compensation.

The muscles of respiration suffer in the majority of cases, and their

impairment constitutes a grave source of danger to life. The intercostals rarely escape altogether; the diaphragm is involved in about one third of the cases, and when it is attacked quickly loses all power, so that the respiration is carried on only by the intercostals, and

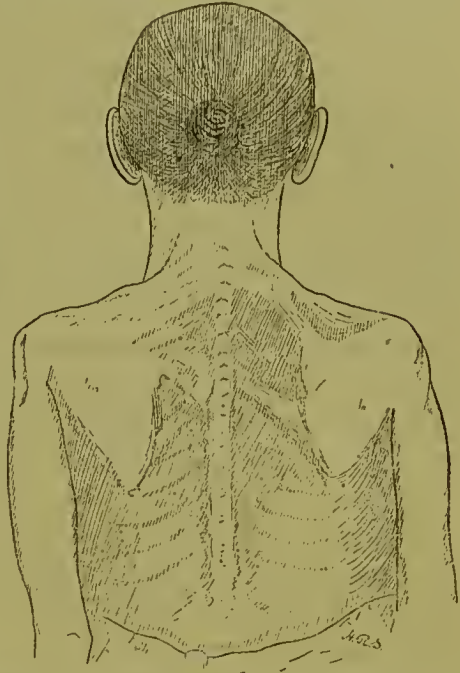


FIG. 127.—Progressive muscular atrophy. Wasting of right trapezius complete (the rhomboids remaining), of the left trapezius partial. On each side the upper part of the trapezius is wasted, and the contour of the neck is correspondingly changed. Both deltoids are also atrophied. (Drawn by Dr. Spencer.)



FIG. 128.—Progressive muscular atrophy. Weakness of the muscles of the neck. (A) habitual posture of the head inclined backwards. (B) position into which it falls if the patient attempts to keep it in the normal balance. (Drawn by Dr. Spencer.)

superior thoracic muscles. On the other hand, the intercostals may suffer much, and the diaphragm remain free; respiration is then purely abdominal and the walls of the thorax may be almost motionless in breathing, or there may be merely a slight movement of elevation of the upper ribs. The chest becomes flattened in front, and narrow from before back, from the influence of atmospheric pressure on the ribs, unopposed by the intercostals. Either the upper or lower intercostals may suffer most, with a resulting difference in the relative degree of movement of the upper or lower parts of the chest. In cases in which the diaphragm is paralysed, the upper chest-muscles often suffer more than the lower; there is then a compensatory over-action of the lower part, and the increased movement of the lower ribs, carrying forwards the abdominal wall, may suggest that the diaphragm is acting when it is not. A careful examination will always prevent the error. The muscles of the abdominal wall occasionally waste, but far less frequently than those of the thorax.

Wasting in the legs is much less common than in the arms, and if it occurs is usually slighter in degree, but occasionally the disease first manifests itself in the legs, and is more intense in them than elsewhere. The glutei, extensors of the knee, and the muscles in the front and on the outer side of the lower leg, are those that are most affected. We shall presently see, however, that the legs are often paralysed when they are not wasted, and sometimes they are the seat of wasting which differs in certain features from that which is the special characteristic of the disease.

The face almost always escapes the general wasting, and its normal appearance may present a striking contrast to the rest of the body. In many cases, however, the lips are paralysed as part of the bulbar palsy that so often accompanies the spinal disease. A remarkable case in which the face shared the atrophy of the limbs, and the tongue escaped, has been recorded by Langer.*

As the wasting progresses, the appearance of the parts in which it is most advanced becomes extremely changed. All trace of muscle may disappear from parts of limbs or even from an entire limb, and there is usually a wasting of the adipose tissue as well as of the muscle, so that the bone seems to be covered only by fascia and skin. The transverse processes of the vertebræ may be felt in the hollow beside the spine, and the bony prominences about the shoulder may be almost as conspicuous as in a skeleton, so that beneath the acromion there may be a groove, into which the finger can be placed, in consequence of the descent of the head of the humerus from the glenoid cavity. The unequal affection of antagonistic muscles leads to various contractions and deformities. In the hand, especially, distortion is apt to occur; from the paralysis of the interossei and the contraction of the longer flexor and extensor muscles, the "claw-like hand" develops in extreme degree (Figs. 17 and 18, p. 30). If all the muscles of a part are

* Meeting of the Vienna Medical Society, March 17th, 1882.

equally and simultaneously affected, no deformity results unless an unsupported part yields to the influence of gravitation. Lordosis is very common in cases in which the trunk- and hip-muscles are involved. Its precise mechanism is not always clear, but it is certainly often the indirect result of the weakness of the extensors of the hip-joint, in consequence of which the pelvis is unduly inclined forwards, and carries with it the lower lumbar vertebræ; the upper part of the trunk has then to be carried far backwards, to bring the centre of gravity within the base of support. The evidence of this mechanism is that the lordosis entirely disappears when the patient sits, and the pelvis, resting on the ischial tuberosities, is no longer inclined. This was the case, for example, in a woman with extreme lordosis from progressive muscular atrophy, in whom, when standing, a plumb line from the seventh cervical spine fell three inches from the sacrum and an inch and a half from the buttock. Sitting, her spine was perfectly straight.

The electrical irritability of the wasted muscles presents changes which vary in character in different cases. When the wasting is slow, there is usually a diminution in both faradaic and voltaic irritability, similar in character to each current. The irritability fails with the muscular nutrition, and when the wasting is great only a slight contraction can be obtained, even with a strong current. When the wasting is extreme, irritability at last becomes extinct, but the voltaic irritability of the muscular fibres persists longer than the faradaic irritability, and a strong voltaic current will often cause a slight contraction long after faradaism, in any strength that can be borne, fails to stimulate the muscle. In such cases, the quality of the voltaic irritability may be normal, but often there is a change in the order of reactions. ACC (anodal closure contraction) occurs as readily, or more readily, than KCC. Tetanic contraction during the passage of the current is produced with undue readiness, compared with the closure contractions, and opening contractions are often caused by currents but little stronger than those that cause closure contractions. Thus there is the qualitative, without the quantitative character of the reaction of degeneration (see p. 45). When the wasting is rapid, and especially when the weakness comes on more rapidly than nutrition fails, there may be considerable diminution of faradaic irritability, while that to voltaism is but little lower than normal, and in such cases the qualitative change is often well marked. Lastly, when there is rapid and considerable paralysis developing in the course of a few days, either at the onset or during the course of the disease, the palsy being followed by rapid wasting, there may be quick extinction of faradaic with actual exaltation of voltaic irritability, and the reaction of degeneration may be present, in all its characteristics.

The mechanical irritability of the muscles is considerably increased; a tap causes a local contraction of the fibres struck. Moreover,

spontaneous flickering contractions of parts of the muscles are very common, now of one bundle, now of another, conspicuous to the eye, although scarcely felt by the patient. This "fibrillation" as it is termed, is so frequent as to be characteristic, but it is not invariable, nor is it confined to this disease. It may sometimes be observed in muscles that are not yet invaded by the wasting, but where it is observed, atrophy usually follows.

In the parts affected by the characteristic wasting all reflex action is abolished, clearly in consequence of the damage to the motor part of the reflex arc; the afferent portion of the arc is unaffected, for, as we shall see, there is no loss of sensation. The myotatic irritability ("tendon-reflex action") is also lost, and lost early. The knee-jerk, for instance, disappears as soon as there is even a slight diminution in the bulk of the thigh-muscles, in cases in which the legs are the seat of primary wasting. The muscles are flaccid and toneless; a condition that may be conveniently termed "atonic atrophy." To this common rule, however, exceptions are sometimes met with; there may be from the first rigidity of the affected muscles. When this is the case, the wasting does not go on to the entire destruction of the muscle; it is often trifling, but may be considerable. With this rigidity there is a preservation of myotatic irritability. We may term this "tonic atrophy." There is thus a marked contrast in the two conditions; we shall presently consider the difference more fully.*

Sensory symptoms are usually slight and always subordinate. They are confined to pain, usually dull and rheumatoid in character, felt chiefly in the limbs in which the malady is most active. Such pains are common at the onset, as we have already seen, and they may recur from time to time during the course of the disease. Sometimes vague feelings of "numbness" or "deadness" are complained of, but cutaneous sensibility is never impaired. (When anæsthesia accompanies muscular wasting both symptoms are due either to chronic meningitis, damaging the nerve-roots, or to disseminated focal myelitis.) Nor do the muscles lose such sensibility as they normally possess. Perception of posture persists, and the muscles are not only sensitive to pressure, but are often more sensitive than in health, not only to pressure but also to extension.

When the arms are the seat of such atrophy as has been described, the legs, if not also wasted, may be normal, but they are often paralysed without being wasted. The loss of power comes on slowly and gradually, and is accompanied by an increase of myotatic irritability. The knee-jerk is excessive, a clonus can readily be obtained, and the

* It should be noted, however, that in the muscles that are the seat of flaccid wasting, when the atrophy has attained an extreme degree, slight rigidity may develop, accompanied by considerable tenderness of the muscle. It is probable that this rigidity is idiopathic, and due to the structural changes in the muscles, presently to be described. This condition, which should be distinguished from the initial rigidity, is rare: the early relaxation is usually maintained to the last.

reflex over-action of the muscles may increase to actual rigidity and spasm, so that there is the condition of spastic paraplegia described in a preceding chapter. It is rare, however, for the spasm to reach the higher degree of intensity, and for the muscles to present the massive firmness that characterises the simple form of spastic paraplegia. On the other hand there is often some diminution in the size of the muscles, and thus we have a gradation to the condition of tonic atrophy just described. In this condition, moreover, there is usually only a moderate diminution in electrical excitability, which does not go on to extinction. The one condition does not seem to pass into the other; the rigidity and myotatic excess does not give place to muscular relaxation; atonic atrophy does not succeed the tonic wasting. In very rare cases, of which I have seen a few instances, there is a similar condition in the hands, when the shoulders are the seat of simple atonic atrophy. The forearms are then rigid, moderately wasted, with myotatic excess, while the muscles of the shoulders are extremely wasted and absolutely flaccid. In still more rare cases, the whole arms are thus affected, are thin and rigid, and in no part is there atonic atrophy.

In one remarkable case of this character the muscles of the trunk participated in the spasm. After coughing or yawning respiration would be checked for a few seconds by general spasm, and whenever the patient was raised from the bed, the arms, legs, back, and neck became stiff in tetanoid rigidity, the head being bent backwards.

The extreme emaciation of the most affected parts shows that the adipose tissue wastes as well as the muscles. The atrophied limbs are usually cold and may be livid or pale, but there is no tendency to acute vaso-motor disturbance in the ordinary form of the disease. I have known the skin of the face to become thin and smooth, so that, in one instance, the dark iris could be seen through the closed eyelids, but such a change is quite exceptional. In another patient there was a very remarkable form of local atrophy. Certain areas of muscles underwent wasting, the rest being normal; the affection commenced by a livid discolouration of the skin, and the wasting seemed to involve the skin, subcutaneous tissue and muscle, causing local depressions. Considerable tracts of the trapezii were thus affected, and smaller spots in the arms and legs. The patient was a single woman of thirty-five, and the disease did not show a strongly progressive tendency.

The functions of the sympathetic are not, as a rule, deranged. Dilatation or contraction of one pupil has been frequently observed, chiefly in association with atrophy of the muscles that are supplied from the lower part of the cervical region, and no doubt depends on the disease of the spinal cord, and not of the sympathetic itself. The reflex action of the iris is usually normal, and optic nerve atrophy never occurs. I have once met with a remarkable reflex fixation of the eyeballs in a case of advanced progressive muscular atrophy. If the patient, looking

to one side, was suddenly told to look at an object on the other side, his head was instantly turned towards the second object, while the eyes remained fixed on the first, by a movement corresponding to that of the head, but in the opposite direction, and then, after a few seconds, they were slowly moved towards the second object. The phenomenon continued to the end of the patient's life.*

The visceral functions are usually little disturbed. Sexual power is often lost. The sphincters rarely suffer. Even when the wasting is general and extreme they may be unaffected, but occasionally they are involved, and they may even suffer early. In other cases, in which the legs become weak after the wasting sets in in the arms, the affection of the sphincters may coincide with that of the legs. In the composition of the urine slight changes have been found, but not constantly. Urea has been increased in some cases, lessened in others.† The quantity of lime has been found to be increased (Fromman), that of kreatin diminished, even to one tenth of the normal (Rosenthal, Langer). The lungs may suffer from the impairment of breathing power, when the intercostals and diaphragm are weakened.

Varieties.—The chief varieties of the disease depend on the relative distribution of the three conditions: (1) atonic atrophy, becoming extreme, (2) muscular weakness with spasm, but without wasting, or with only slight wasting, and (3) tonic atrophy, rarely extreme in degree, with myotatic excess. The commonest condition is to have atonic atrophy in the arms and upper part of the trunk, with simple weakness and spasm in the legs. Atonic atrophy in both arms and legs is less common, and the least common is tonic atrophy alone, in the arms, or universal. The last is, indeed, extremely rare. It is important to note, however, that these conditions may coexist in every degree and combination; between universal atonic atrophy on the one hand, and universal spastic paralysis without wasting on the other, there is every gradation. The latter does not come into the category now under consideration, but similar cases are met with in which there is atrophy of a few muscles, as, for instance, of the hands only, which complete the series.

Complications.—Progressive muscular atrophy is occasionally accompanied by the symptoms of some other degenerative disease of the spinal cord. The paralysis with spasm, already described, can scarcely be regarded as a complication; it is rather part of the disease, and its relation to the other symptoms will be considered when we discuss the pathology of the affection. In rare cases muscular atrophy in the arms is accompanied by the symptoms of locomotor ataxy in

* As I pointed out in an account of this curious condition ('Brain,' vol. i) it is interesting evidence of a normal reflex mechanism in the fixation of the eyes, which was, as it were, isolated by disease, which lessened voluntary control over it.

† In a patient who weighed 7 st. 13 lbs., and whose urine varied between 760 and 960 cubic centimetres, I found the daily excretion of urea to vary between 10·7 and 15 grammes, the average being 13 grammes. This is just half the normal average for a man of that weight, which would be (according to Parkes), 24 grammes.

the legs. By far the most frequent complication is bulbar paralysis, weakness of the lips, tongue, pharynx, and often of the laryngeal muscles. It is the expression of a degenerative process in the medulla, similar to that which, in the spinal cord, causes the affection of the limbs. It may come on at any stage of the disease, may precede the spinal symptoms, or only come on when these have attained a considerable degree. In characters, the bulbar palsy may resemble perfectly that which occurs in isolated form and will be described among the diseases of the brain. Frequently, however, there is but little paralysis of the tongue, even when swallowing and articulation are much impaired. There may also be slight interference with articulation when there is no marked bulbar palsy. A minor complication is the neuralgic pain, which, as already stated, is sometimes troublesome in the early stage of cases that are due to cold. In several patients I have known headache to be frequent and severe throughout the course of the disease.

Course.—The malady, in most cases, is steadily progressive, as its name implies, but in the rate of its progress it varies in different cases, and in the same case at different periods. It may, moreover, become stationary at any period in its course, and when once actually arrested, it does not usually again become active. Unfortunately, the tendency to cessation is greatest in the later stages of the disease, when there is little except life to be preserved. Sometimes progress ceases at an earlier stage, and chiefly, I think, in those cases in which the atrophy is strictly symmetrical, and develops simultaneously, or almost simultaneously, on the two sides. Thus I have met with several cases in which there was symmetrical wasting in the two hands, or in certain muscles of the two forearms or of the two upper arms, and in which the atrophy, after attaining a considerable degree in its limited seat, had become stationary and continued so. Occasionally some accessory symptom may pass away, although the atrophy progresses. Thus I have known power over the sphincters, at first lost, to be regained in spite of the steady advance of other symptoms.

When the progress at the commencement is rapid, it usually continues rapid, until the disease has attained a wide extent. When it begins slowly, it may continue to be slow throughout, or may, after a time, become accelerated.

Although the disease sometimes commences in the second arm very soon after its onset, it more commonly makes some progress in its primary seat before beginning on the second side, and the interval that elapses varies with the rate of extension. It often happens that a year intervenes between the affection of the two arms, and I have known, in a very chronic case, the atrophy to slowly progress in one arm for seven years before it showed itself in the other. It is not common for the arms to be reduced to practical helplessness in less than two or three years, but the hands may become useless in as short a time as six months, while in one case the wasting, commencing at the

shoulder, had invaded the whole arm in the course of a month. The shortest time in which I have known a patient to reach the last stage of the disease is nine months.

With any rate of general progress, the otherwise uniform course of the disease may be broken by the occurrence of almost sudden palsy in a certain group of muscles. Considerable loss of power, it may be absolute paralysis, comes on in a few days, or even in a few hours. It may occur at the onset of the affection. The extensors of the wrist and fingers are the muscles most commonly thus affected. The weakness is usually followed by a well-marked degenerative reaction in the muscles. In one case, which began by such subacute paralysis of the extensor muscles, first in one arm and then in the other, the initial condition closely resembled the paralysis from lead-poisoning. This cause was, however, excluded, and soon the muscles of the shoulder and back presented commencing slow progressive atrophy. In another case the quick loss of power was confined to the extensors in one arm, which were already weak and slightly wasted, most of the other muscles of the upper limbs having been long atrophied. When there is weakness of the legs, without wasting, the onset of this may coincide with the atrophy of the arms, or may succeed it at any interval. In one case five years elapsed, after the arms began to waste, before the legs became weak. It is very rare for the paraplegic weakness to occur first.

The chief danger to life is from pulmonary maladies, rendered grave from the weakness of the muscles of respiration. The common complication of bulbar paralysis is another frequent cause of death, either by the interference with swallowing and nutrition or by the laryngeal paralysis. Less commonly death results from bedsores and septicæmia, or from intercurrent maladies.

PATHOLOGICAL ANATOMY.—The wasting of the muscles is as evident after death as during life. They are reduced in size and pale in colour. Sometimes there is little in the tint of what remains to suggest muscular tissue. Parts of a muscle may be hardly distinguishable from adjacent fat. On the other hand the bulk of the muscle may be dark, and pale streaks in it may mark the position of local degeneration. Under the microscope the fibres present various changes, and of these four are well defined. (1) There may be simple narrowing of the fibres, without any considerable change in their striation (Fig. 129) although the striæ often seem to be further apart than normal, and sometimes the fibrillary segmentation is unusually distinct. (2) Simple fatty degeneration, in which the transverse striation gives place to a granular appearance (Fig. 130), the granules become larger and fewer (Fig. 129) until ultimately distinct globules are scattered through the sheath. Where the muscle resembles fatty tissue to the naked eye, the microscope may show only sarcolemma sheaths containing groups of globules. (3) Muscular fibres are seen in which the sheath contains only a clear material enclosing a few fatty

globules, and a few transverse striæ, faint, as if fading away. It is probable that this is not the result of fatty degeneration but of a different process, which has been termed "vitreous degeneration,"—a sort of dissolution of the striæ, indicated by the appearance of such fibres as are shown in Figs. 129, 131, 132. (4) A longitudinal striation develops in the fibre, and at first co-exists with the trans-

FIG. 129.

FIG. 130.

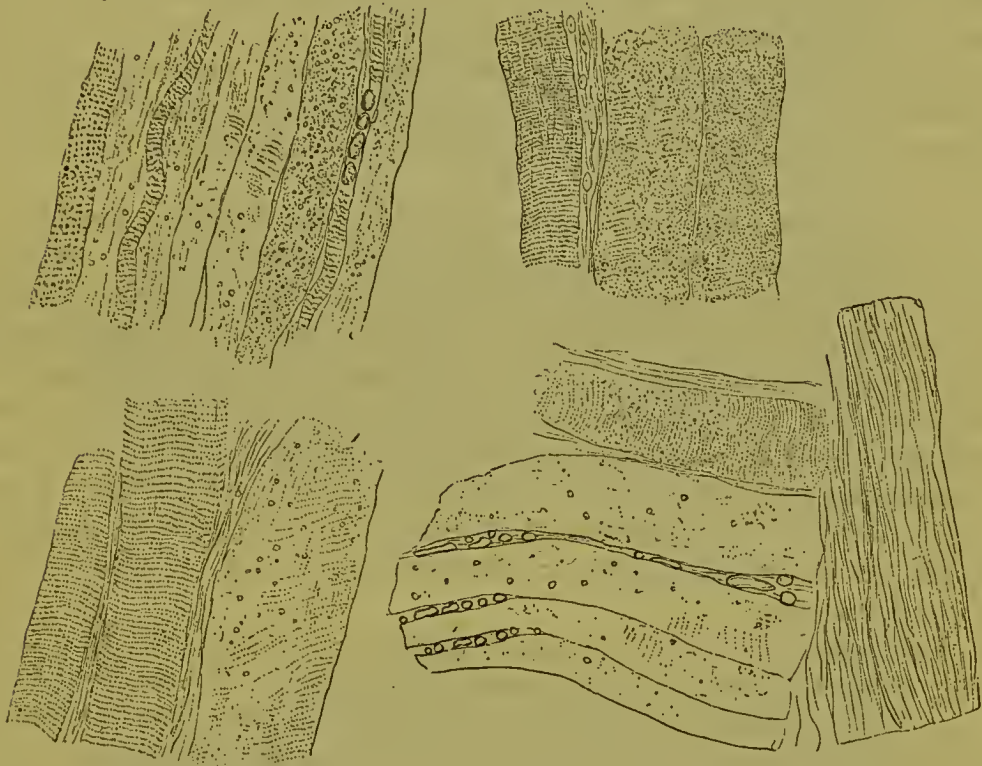


FIG. 131.

FIG. 132.

FIBRES OF WASTED MUSCLES IN PROGRESSIVE MUSCULAR ATROPHY.

FIG. 129.—Narrowed fibres with nearly normal striation: others clear, containing a few fat globules, and a few faint transverse striæ, and some longitudinal striation: large globules of fat lie in the interstitial tissue in front of one narrowed fibre.

FIG. 130.—Granular degeneration: a fibre with normal transverse striation presents also indications of longitudinal striation. Increase of nuclei of interstitial tissue.

FIG. 131.—Two normal fibres and one presenting the clear homogeneous aspect with a few fat globules and some faint striation.

FIG. 132.—Several fibres similar to that of the last figure, with globules of fat between them. On the right is a fibre which has undergone complete longitudinal striation, the normal striæ having disappeared, so that it resembles the adjacent interstitial fibrous tissue. Other fibres in the same muscle were in intermediate stages of degeneration, some transverse striation coexisting with the longitudinal striation.

verse striation, but ultimately the latter becomes indistinct, and the fibre looks like a fasciculus of longitudinal connective-tissue fibres (Fig. 132). Sometimes, with the longitudinal striation, the fibre presents a transverse striation very much finer than normal, the striæ being narrower and nearer together as if from a division of the 'sarcous elements.' This change may sometimes be seen alone, and

may be present in only one part of a fasciculus. Thus at one place the number of striæ in $\frac{1}{1000}$ inch was only seven, while in another region of the same fasciculus seventeen were to be counted in the same space. A tendency to transverse fissuring has also been described, but this is probably artificial. Fatty globules accumulate between the fibres (Fig. 132) accompanied in some cases with granules and masses of reddish-brown pigment. There is often also an increase of the nuclei (Fig. 130) and sometimes of the fibres of the interstitial tissue. Two or three rows of nuclei may lie between the fibres. The capillaries may be dilated and distended. It is very common to see muscular fibres that are much altered side by side with others that present a nearly normal appearance. Ultimately the sheaths become empty, and shrink, and may be scarcely distinguishable from the interstitial fibrous tissue.

The peripheral nerves contain many degenerated nerve-fibres, and the terminal branches for the muscles a still larger number. If the nerves are traced up to the cord it is found that the degenerated fibres come only from the anterior roots. These are conspicuously changed to the naked eye, small and grey. They may resemble fine threads of connective tissue, and under the microscope no nerve-fibres may be found in them, or only a few may remain of normal aspect, the rest being in various stages of degeneration or represented only by their empty sheaths. The degree of affection of the anterior roots corresponds to the wasting in the parts supplied by them. The posterior roots are normal.

The spinal cord is often softer than natural at the affected part, and the white substance of the lateral columns may be conspicuously translucent in aspect, especially in the cervical enlargement. Under the microscope morbid changes are seen in the anterior cornua, and also, in most cases, perhaps in all, in the antero-lateral white columns. The change in the anterior cornua corresponds in its intensity to the origin of the nerves to the most affected parts, and since the latter are usually the arms, the disease is generally most intense in the cervical enlargement. In stained sections the grey matter of the horn is less deeply tinted than normal, but in some parts it may stain more deeply, especially in the circumference of the cornu and the processes of grey matter which project into the white column. Occasionally larger areas have a dense aspect and stain deeply, but the central part of the cornu is generally pale and more translucent than normal. Most of the large nerve-cells have disappeared; many entirely, while others may be represented only by small angular bodies. In many instances not a single large cell can be seen in a section which, in a normal cord, would contain a large number. Frequently, however, a few large cells can still be seen, but most of these have lost their processes and are more globular than normal. The interstitial tissue is also changed. The nerve fibrillæ waste with the cells, and there is an increase of the small, angular, and stellate cells and other connective-

tissue elements. The larger vessels are dilated and surrounded by wider spaces than normal, but there is no considerable distension of the capillaries. The cornu as a whole is not usually changed either in size or shape. Similar alterations may be traced through the dorsal

FIG. 133.

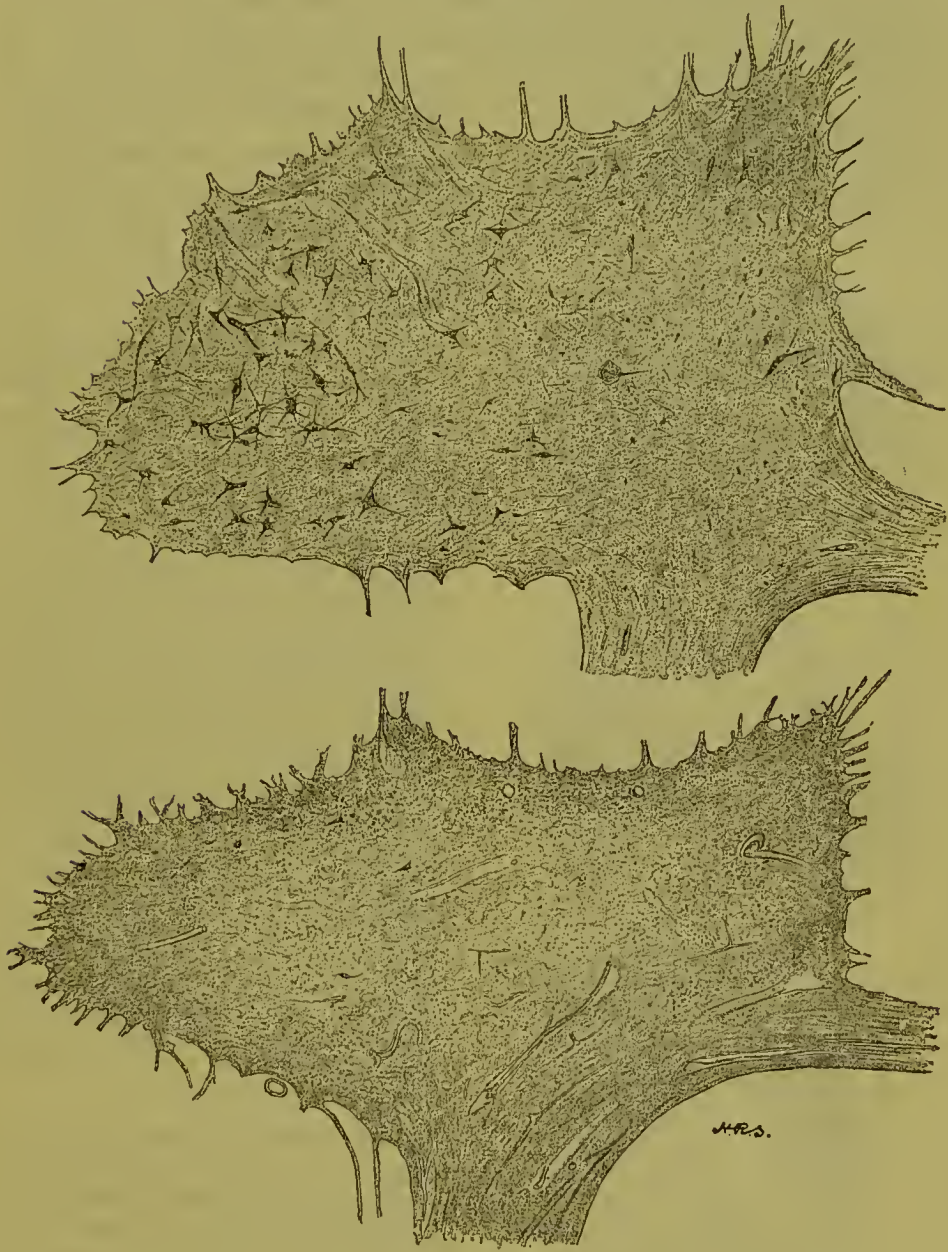


FIG. 134.

FIG. 133.—A normal anterior cornu for comparison with the next fig.

FIG. 134.—Progressive muscular atrophy; anterior cornu, cervical region.

(From drawings by Dr. H. R. Spencer.)

region. In the lumbar enlargement, the grey matter may be normal, even when it is much altered in the cervical region, but if the legs are wasted there are changes similar to those already described. When



the atrophy begins in the legs, the disease may be more intense in the lumbar than in the cervical enlargement; but more often the degeneration is slighter, and a larger number of normal or slightly changed cells can be seen. Sometimes certain groups of cells are little affected while others are much atrophied. The degeneration may involve to some extent the intermediate grey matter between the cornua, but here it ceases; the posterior horn is always normal.

There is distinct degeneration of the anterior root-fibres pass-

FIG. 135.—Progressive muscular atrophy; degeneration of the anterior cornua and pyramidal tracts. A, medulla oblongata, complete degeneration of the anterior pyramids A P; B, at the upper part of the decussation of the pyramids; D, the decussation of the degenerated fibres; A P, the pyramids, still incomplete. C, cervical, D, dorsal, E, lumbar sections. The degeneration of the anterior cornua is complete in C, but in E a few cells remain, for the most part without processes. In C and D the degeneration of both anterior and lateral pyramidal tracts is conspicuous; in E the anterior tract has ceased, and the lateral tract extends up to the surface of the cord, from which it is separated in C and D by the undegenerated direct cerebellar tract. In C and D the degeneration extends forwards in front of the lateral pyramidal tract, but gradually ceases. The fibres of the anterior commissure are also degenerated.*

* In this case the arms were greatly wasted and flaccid; the legs were paralysed, rigid, with moderate wasting. The muscles of the back were also atrophied. In the arms the weakness and wasting came on at the same time, in the right arm some time before the left. The shoulder-muscles suffered first, but the atrophy quickly spread, and was ultimately extreme in the muscles of the shoulders, forearms, and hands. The disease ran a rapid course; death occurred two years after the gradual onset.

ing from the cornu through the anterior column. A few fibres may remain, but whole fasciculi appear to be replaced by fibrous tissue. There is also degeneration of the fibres of the anterior commissure, in consequence of which it stains much more deeply than normal, in the regions in which the grey matter is considerably diseased.

In the white columns there is usually considerable and often almost complete degeneration of the pyramidal tracts, anterior and lateral.* The area of sclerosis, resulting from the degeneration of the two tracts, varies in extent according to the size of the anterior tract and

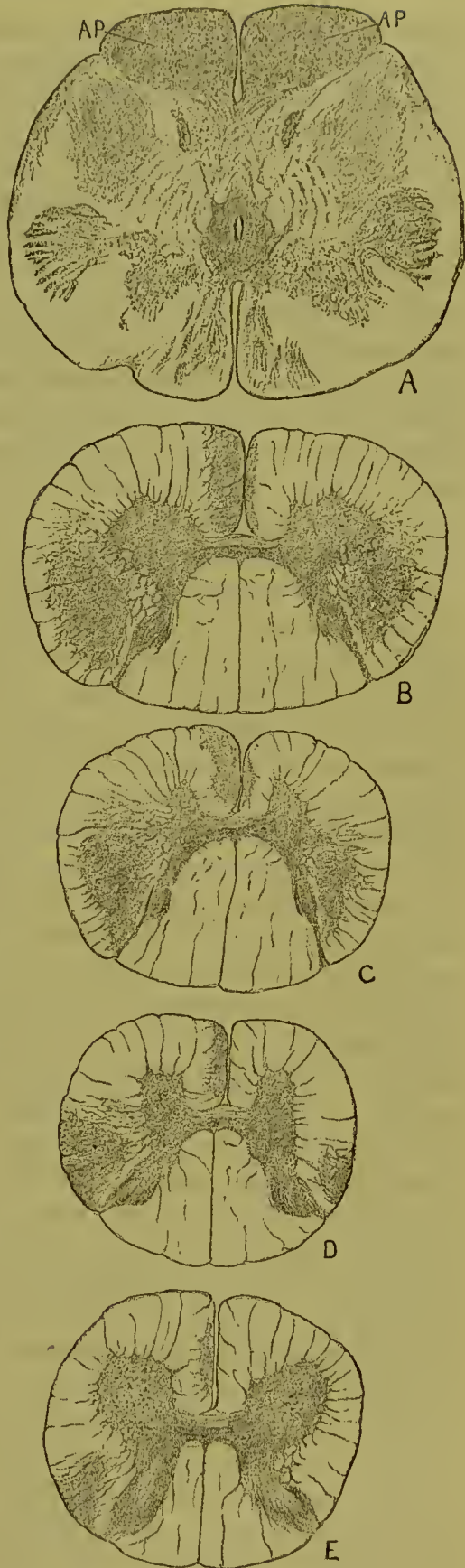


FIG. 136.—Progressive muscular atrophy. A, medulla oblongata; degeneration of the anterior pyramids A P. B, cervical, C, first dorsal, D, first lumbar, E, mid-lumbar sections. The nerve-cells have disappeared from the anterior cornua, except in E, in which a few remain. There is complete degeneration of the pyramidal tracts, anterior and lateral. The decussation at the medulla was unequal in this case; of the left pyramid fewer fibres crossed than of the right, and hence the left anterior pyramidal tract is larger, while the right lateral tract is smaller than normal, and, moreover, has ceased at the middle of the lumbar enlargement, to which the left anterior tract extends.†

* I have not yet met with a single case of progressive muscular atrophy in which the pyramidal tracts were unaffected, and I am not aware that any case of the kind has been published since attention was directed to the affection of these tracts by the researches of Charcot.

† The patient was a man forty-eight years of age. The disease followed a severe exposure to cold. The wasting of the arms was extreme, and they were absolutely powerless. The legs were much less wasted than the arms, but very weak, the loss of power being out of proportion to the wasting.

to the distance which it extends down the cord. In Fig. 136 this is illustrated in an unusual manner in consequence of an inequality in the decussation of the pyramids at the medulla, fewer fibres of the left pyramid having crossed than of the right, so that the left half of the cord contains more than its proper share of the fibres, and the anterior tract extends into the lumbar region even further than does the lateral tract. Where the direct cerebellar tract exists, this, always unaffected, limits the sclerosis of the lateral tract on the outer side. On the inner side, the "lateral limiting layer" (see p. 122) is usually much less degenerated than the pyramidal tract, and intervenes between the latter and the grey matter, but this layer suffers in some degree. The sclerosis does not cease at the anterior extremity of the pyramidal tract, but extends forward in the "mixed zone" of the lateral column, lessening in intensity, and ceasing usually opposite the outer part of the anterior cornu. It is here most intense close to the grey substance, and ceases before it reaches the surface of the cord, the antero-lateral ascending tract being unaffected. It is probable that this sclerosis is due to the degeneration of the short vertical fibres that pass between adjacent regions of the anterior cornu, and which share the degeneration of the nerve-cells. Occasionally this sclerosis extends, in slighter degree, into the anterior columns. The posterior columns are always free from definite degeneration, but occasionally there is some general increase of connective tissue throughout the whole cord. The degeneration of the pyramidal tracts is usually considerable in degree, and often most of their fibres seem to have perished. If it is incomplete in the cervical region it may be slight in the lower part of the cord, the degenerated fibres being chiefly those that end in the cervical enlargement and dorsal region. When the sclerosis is intense it is not confined to the cord; it can be traced up through the decussation (Fig. 135, B) and through the medulla oblongata, in which, as Figs 135 and 136 show, the whole of both pyramids may be degenerated, so that they stain deeply, and no nerve-fibres can be distinguished in them. The degeneration of the pyramids has been found in several cases to extend through the pons and crus to the internal capsule, and even through the white substance to the cortex. Above the capsule, however, the pyramidal fibres are so mingled with others that their degeneration is no longer recognisable by the deeper staining of a definite tract; but it can be detected by the presence of abundant products of degeneration in the path of the fibres. In the motor cortex, the large ganglion cells have been found fewer than normal, and distinctly degenerated, many having lost their processes, while in the interstitial tissue, spider cells and other connective-tissue elements are increased in number.*

In cases in which the symptoms of bulbar paralysis were present during life, the motor nuclei of the medulla have presented changes

* Koschewnikoff, 'Archives de Neurologie,' 1883, No. 18; and 'Centralblatt f. Nerven-krankheiten,' 1885, p. 409; Marie, 'Neurologisches Centralbl.,' 1884, p. 61.

corresponding to those of the grey matter of the spinal cord, with a similar degeneration of the nerve-cells. In other instances the degeneration of these nuclei has been slight, but in such cases the degeneration of the pyramids has been intense, and doubtless involved the fibres connecting these nuclei with the cortex of the brain.

The sympathetic nerves and ganglia, when examined, have been found normal, or have only presented such changes as are common apart from symptoms of disease of the nervous system.

PATHOLOGY.—The theories according to which the disease was regarded as primarily one of the muscles or of the sympathetic nerves have now only an historical interest. The constancy of the changes in the ganglion cells of the spinal cord, the degeneration of the motor root-fibres, and the analogous effects of acute lesions of the anterior cornua, leave no doubt of the relation of the muscular wasting to the disease of the grey matter, and essentially to that of the ganglion cells. The slowness of the change causes the impairment of the nutrition of the muscular fibres to proceed *pari passu* with that of the nerve-elements, and the interference with motor conduction to be proportioned to both. We are thus able to understand the condition of electrical excitability, and its slow failure as nerve and muscle degenerate together. It is only when the slow degeneration is varied by a more acute process of destruction of cells and degeneration of fibres that the muscular tissue is for a time less damaged than the nerve-fibres, and presents paralysis in excess of the wasting, and a voltaic irritability in excess of the faradaic irritability of the nerve-endings (p. 20). Thus the essential lesion of the disease is a slow decay of the lower segment of the motor path, the segment which consists of the ganglion cells and their prolongations in the axis-cylinders of the nerve-fibres (see p. 116). To this the conspicuous lesion, the muscular wasting, is secondary. It is perhaps better thought of as a degeneration of the whole segment than as simply a lesion of the ganglion cells, although the latter being the element on which the nutrition of the segment depends, the two views are merely different modes of stating the same fact.

But the disease is rarely limited to the lower segment of the motor path. We have seen that the pyramidal tracts are commonly degenerated, and it is probable that the degeneration often extends through their entire extent, and involves the motor cells of the cortex, as it has been proved to do in several cases.* Hence the upper segment is often degenerated as well as the lower segment. In the presence of that complete degeneration of the lower segment which causes the atonic atrophy of the muscles, the degeneration of the upper segment seems to cause no symptoms. The loss of power that it would produce

* At the same time we have seen that the nutritional stability of the fibres is less in the lower than in the upper parts, and that their isolated degeneration may be greatest in the lower parts. This may sometimes also be the case in progressive muscular atrophy.

is also caused by the degeneration of the lower segment, and the latter abolishes the myotatic irritability, excess of which is the characteristic indication of disease of the upper segment. Hence the degeneration of the upper segment of the motor path to the muscles that present atonic atrophy cannot be recognised during life.

What is the relation between the degeneration of the two segments, between the lateral sclerosis and the affection of the ganglion cells? We cannot assume (as some have been inclined to do) that the affection of the upper segment is secondary to that of the lower, because a primary lesion of the grey matter, such as occurs in cornual myelitis, does not cause any pronounced ascending degeneration of the related pyramidal fibres, although they may undergo slight degeneration in the course of many years, apparently from disease. Even a complete interruption of the pyramidal tracts is followed by no degeneration of their upper parts. Hence the intense degeneration met with in progressive muscular atrophy cannot be regarded as secondary and ascending. Neither, when there is atonic atrophy, can we consider the affection of the upper segment to be the primary lesion, and to be the cause of that in the lower. In the early stage of such atrophy there are no indications of the degeneration of the upper segment, which would then certainly exist if such degeneration preceded that of the lower segment. Moreover, degeneration of the upper segment does not necessarily cause any degeneration of the lower. It is a matter of every-day observation that intense degeneration of the termination of the upper segment may occur, from dorsal myelitis, for instance, without any considerable wasting of the legs, and such degeneration has never been known to excite the complete degeneration of the lower segment which causes atonic atrophy. Hence the only adequate explanation of the facts is that the degeneration of the upper and lower segments is simultaneous, or if not simultaneous, at least so far independent that neither is the cause or consequence of the other; both are the result of the same tendency to degeneration of the motor path. Atonic muscular atrophy is thus, at least in many cases, the visible expression of a tendency to decay of the whole motor path from the cortex of the brain to the muscles.

The simple weakness of the legs, with excessive myotatic irritability and spasm, is explained by the degeneration of the pyramidal fibres for the legs, the lower segment being unaffected. In such a case the nerve-cells of the lumbar enlargement are normal. When this condition is associated with slight wasting of the legs, without considerable change in electrical irritability, the condition exists that we have more than once considered, in which we must assume that the motor nerve-cells of the cord, while structurally intact, undergo slight changes in nutrition. In this condition there are many nerve-cells of normal appearance in the grey matter. The changes in nutrition are too slight to cause changes in aspect, or if they cause such changes we have not yet learned to identify them. They are perhaps results of the degenerative changes

in the termination of the upper segment, but when such degeneration is secondary to a focal lesion of the cord or brain, the changes in nutrition of the cells never attain such a degree as to arrest the myotatic irritability, or to cause wasting such as attends the destruction of these cells.

In some cases, however, the muscular wasting may be great, although the increase of myotatic irritability persists. In these cases, as we have seen, there is considerable rigidity of the muscles throughout the whole course of their wasting, the condition that we have termed "tonic atrophy." In such a condition it is common to find that many nerve-cells have disappeared or are very small, but others remain normal or slightly changed in aspect. Apparently, in addition to the degeneration of the upper segment and to the nutritional changes just mentioned, we have then a considerable degeneration of many, but not destruction of all, the elements of the lower segment. We cannot regard this as simply secondary to the degeneration of the upper segment, for the reasons already given. It must be the expression of a distinct pathological tendency similar to that which elsewhere causes the atonic atrophy and total wasting but slighter in extent and later in time; insufficient to prevent the less affected cells from causing rigidity under the influence of the degeneration of the upper segment. It is doubtful whether the tonic atrophy ever goes on to atonic atrophy. Theoretically conceivable, it is certain that if it ever occurs it is extremely rare. Nor does it seem that atonic atrophy ever gives place to tonic atrophy, with excessive myotatic irritability. The rigidity of tonic atrophy is due to the degeneration of the upper segment, but the effect is not produced if the lower segment is already the seat of such extensive degenerative changes as abolish myotatic irritability. The pyramidal fibres for the parts that are the seat of atonic atrophy are constantly found degenerated, although the muscles have been flaccid to the last. In the very rare cases in which the muscles, relaxed during the process of atrophy, become rigid towards the end of the process, it is probable that the rigidity is idiopathic, due to the changes in the muscles, and is not dependent on the central nervous system. It may be the result of the increase in the interstitial connective tissue, and the longitudinal division and fibrillation by which the muscular fasciculi come to resemble bundles of connective-tissue fibres. The rigidity is always attended by great sensitiveness of the muscles. It is also possible that similar idiopathic muscular changes may ultimately, in tonic atrophy, maintain and increase the rigidity that is primarily dependent upon the spinal cord.

It has been mentioned that the cases in which the legs present the simple palsy and spasm, or the tonic atrophy, which indicate degeneration of the pyramidal tracts, have been separated by Charcot, and termed "amyotrophic lateral sclerosis," the separation being based on the assumption that in such cases the primary lesion is the degeneration of the pyramidal tracts, and that the affection of the grey

matter is secondary or "deuteropathic," even where the atrophy is atonic. We have seen also that this assumption is unwarranted so far as the atonic atrophy is concerned. It is probable that the pyramidal tracts are degenerated, if not constantly, at any rate in such a very large proportion of the cases of progressive muscular atrophy that Charcot's distinction is in effect giving a new name to an old disease. Whether there are indications of lateral sclerosis or not, depends on the circumstance whether the degeneration of the pyramidal fibres is or is not more extensive than the complete degeneration of the nerve-cells that causes atonic atrophy. If the latter is universal, the pyramidal tracts may be totally degenerated, and yet there may be none of the characteristic indications of such degeneration. On the other hand, both arms and legs may be the seat of the spastic paralysis that indicates pyramidal degeneration, and atonic atrophy may be limited to a few muscles of the hands. Between these we have every gradation of degree and distribution of atonic atrophy, spastic paralysis, and tonic wasting. Of these only the last-named indicates an affection of the grey matter secondary, in point of time, to the pyramidal degeneration, and even then only in part secondary in point of causation. Hence a division into two classes (into which the same case may fall at different periods) is less in harmony with the facts of disease, than is a recognition of the varying extent of the lesion and the corresponding variation of clinical character and course.

The process in the grey matter has been regarded by Charcot and others as a chronic inflammation. The occasional rapid increase in the symptoms may be thought to be justification for this view; but the process in general is at the degenerative extremity of the series of nerve-lesions. The principle involved in this question has been already discussed, and we have seen that, whatever be the nature of the primary process, we must recognise secondary tissue changes of independent energy and that a distinct process of inflammation may occasionally form part of these (see p. 286). The significance of the occurrence of inflammation may therefore easily be overrated, so far as concerns the question of the tissue in which the disease begins.

DIAGNOSIS.—The simultaneous and gradual onset of weakness and wasting, the slow but progressive increase and extension of the symptoms, render the diagnosis of the developed malady simple and easy. At the onset, when only a single muscle or group of muscles is affected, the question arises whether the atrophy is local or is the commencement of a wider affection. If there is no indication of a peripheral nerve-lesion, local atrophy only occurs from great overuse of a muscle, commonly one of the small muscles of the hand, from some manual occupation, and in a person of feeble general strength. If such cause cannot be traced, the wasting must be looked on with grave suspicion, and a careful and repeated examination will, in most cases, soon show some extension of the atrophy.

In multiple neuritis, the onset of the symptoms is subacute, wasting follows weakness, and the danger of confusion is with subacute myelitis of the cornua, not with their slow degeneration. In pachymeningitis of the cervical region, with considerable damage to the nerve-roots, the wasting in the arms may resemble that of progressive muscular atrophy, and there are often weakness and rigidity of the legs, but the wasting is less chronic in onset, and is always accompanied by distinctive sensory symptoms,—by more acute pains, and by anæsthesia, irregular in distribution. The same distinctions suffice for the diagnosis in diseases of the nerve-roots of the cauda equina, as by a tumour. This may cause slow wasting in the legs, but there is always severe pain and loss of sensibility.

From primary muscular atrophy, “idiopathic atrophy,” we may term it, the diagnosis is sometimes easy, sometimes very difficult. It is easy in pseudo-hypertrophic paralysis, even in the cases of this disease in which no muscles are large, on account of its characteristic distribution, its course, the age at which it begins, and the tendency to affect many members of a family, and males more than females. Whenever several cases of muscular atrophy occur in a family, during childhood or youth, the probability is great that they are idiopathic, and not spinal. But cases are sometimes met with in which idiopathic atrophy occurs in adult life and has not the course or aspect of the pseudo-hypertrophic form. The distinction of such cases may be very difficult. The only general rule that can be given is that they run a more chronic course than the spinal form, often slowly progressing during fifteen or twenty years; that their distribution is unlike that of the spinal form; that the affection often involves the face; that the hand-muscles escape in most cases; and that such disease often occurs in many members of the same family. The distinctions will be better understood after a perusal of the account of these idiopathic atrophies.

PROGNOSIS.—The progressive character of the disease renders the prognosis, in every case, unfavorable. The only guide is the observed tendency of the disease. As long as it is distinctly increasing, and in proportion to the rate of increase, the prognosis is grave. Although a hope of arrest is justified, such arrest cannot, in any case, be actually anticipated until it occurs, if the expression may be allowed. Some increase of atrophy in the parts already affected is compatible with an arrest of extension, the muscular changes increasing up to the extent of the disease in the nerve-structures. The prospect of early arrest is greatest in the cases in which the wasting is strictly symmetrical and nearly simultaneous on the two sides. The danger to life is chiefly proportioned to the interference with the muscles of respiration, and to the indications of implication of the medulla. If the malady ceases to advance, the prospect of any recovery depends on the rate at which the disease has progressed. Recent rapid loss of power may be to some extent recovered from, especially when the muscles present

the degenerative reaction. Wasting that has existed for six months will probably persist unchanged. In a typical chronic case there is little hope of any actual recovery of tissue or power. The effects depend on a slow destruction of nerve-elements which are not be regenerated.

TREATMENT.—The first important element is to secure favorable conditions of life, and to maintain the general health as perfect as possible. Fresh air, and gentle exercise are important, but all fatiguing exertion should be avoided, and likewise all mental strain. When the patient becomes helpless, great care is necessary. Bedsores in this disease mean inattention and may always be avoided. Drugs often fail to influence the malady; those that occasionally seem to do some good are the agents that are most frequently useful in other degenerative diseases of the spinal cord. They have been described in the account of the treatment of locomotor ataxy. Among them arsenic and strychnine are certainly most useful. Strychnia injected beneath the skin has certainly more influence over the disease than when given by the mouth. I have known, more than once, the progress of the disease to cease under the influence of injections, although strychnia had been given by the mouth without effect. The nitrate of strychnia is the best salt for hypodermic use, and the injection may be made once a day, beginning with $\frac{1}{80}$ th and gradually increasing to $\frac{1}{40}$ th. The dose should not be increased beyond this. The locality is unimportant. It is possible that, since the agent enters the blood far more rapidly when injected beneath the skin than when absorbed from the stomach, the momentum of its effect on the nutrition of the nerve-elements may be greater.

Local treatment of the muscles has very little influence on the malady, as may, indeed, be expected from its nature. The most sedulous and skilful use of electricity, voltaic or faradaic, fails as a rule to produce any effect on the course of the disease. If the malady is progressing at the same rate in each arm, and the muscles of one arm are regularly treated with electricity, and those in the other arm are left alone, no difference can be detected in the rate of wasting on the two sides. It is possible, nevertheless, that electricity sometimes does a little good. In cases in which a rapid loss of power has occurred, and weakness is out of proportion to the wasting, some recovery is possible, and there is no doubt that the excitability of the muscular tissue is maintained for a longer time by galvanism, although the bulk of the muscle may not be influenced. In other cases all that can be said is that the influence of electricity, properly applied, is in the right direction. Moreover, the disease is one of those in which patients find it hard to believe that electricity cannot help them, and the probability is that their conviction will be fostered by some medical adviser. It is one of the diseases in which unjustifiable assertions are too often made that early electrical treatment would have been successful. It is

often well, therefore, if only to satisfy the patient that nothing has been left untried, that a careful course of electrical treatment should be adopted. Faradism may be used, if the muscles are sensitive to it, but if they present any greater irritability to voltaism, it is better to use this. It is immaterial whether the application is confined to the muscles or whether one electrode is placed over the affected part of the spinal cord. The latter method has no disadvantages, but my own observations have failed to confirm the confident statements sometimes made regarding its superiority. It is very important that the current-strength employed should be moderate. Strong applications often cause much subsequent pain, and even increased disability, and should be carefully avoided. I have known a rapid increase of weakness follow a strong application, in a way that convinced the patient at least that the two were connected.

Rubbing and massage of the muscles have been frequently employed, and of this treatment also it may be said that its influence is in the right direction although usually inappreciable so far as the muscular atrophy is concerned. Combined with passive movement, the influence of rubbing in preventing and diminishing deformities is more distinct. No special bath treatment is of service. When the disease occurs in the subjects of syphilis, specific treatment invariably fails, and I have even known the progress of the disease to be distinctly accelerated by an energetic course of such treatment by iodide and by mercury. It is important that all treatment should be pursued in moderation and that its effects should be carefully watched. The disease is one in which it is not easy to do good and not difficult to do harm.

ARTHRITIC MUSCULAR ATROPHY.

Inflammation of a joint is almost invariably attended with rapid wasting of the muscles that move the joint. This occurs equally whatever be the cause of the inflammation, whether this is spontaneous or traumatic. It attends chronic as well as acute inflammations, and occurs in animals if joint inflammation is produced in them.*

The muscles that waste are chiefly those which extend the affected joint. The atrophy is well seen in the muscles in front of the thigh, when the knee is inflamed. If the ankle is affected, the calf-muscles chiefly waste; if the hip, the glutei; if the wrist, the extensor muscles of the forearm; the triceps when the elbow is affected; the deltoid when the shoulder-joint is inflamed. In rheumatoid arthritis of the finger-joints, such wasting is usually very conspicuous in the interossei and especially in the abductor indicis. The wasting, however, sometimes

* Valtat, 'Archives Générales,' 1877, tome 30, pp. 159 and 321. The subject has been discussed by Vulpian ('Leçons sur l'App. Vaso-moteur,' 1075, t. ii), and by Paget ('Lancet,' 1873, vol. ii, p. 727, in a lecture republished in 'Clinical Lectures and Essays,' 1875, p. 208).

involves the flexors as well as the extensors, and rarely muscles of the limb that are near but do not move the affected joint. In very rare cases all the muscles of a limb have presented some wasting. Cases in which the atrophy is unusual in distribution, and especially when it is on the distal side of the affected joint, must be regarded with some suspicion, because inflammation sometimes spreads from a joint to a nerve, and distant wasting may be thus produced. It is said that there is sometimes, at the onset, a considerable weakening of the muscle, interfering with movement more than can be accounted for by the pain in the joint which movement causes, and that such initial palsy is transient (Valtat, Duchenne). But the pain, by its inhibitory influence, usually obscures such palsy, which is indeed confessedly rare.

The atrophy, on the other hand, may be regarded as almost constant. If the onset of the joint affection is acute the wasting occurs with rapidity. In a day or two the muscles may be flabby, and in a week or ten days a difference in the circumference of the limb may be detected by measurement. However long the affected muscle may be, the wasting involves the whole length of it. It is not limited to the part in the neighbourhood of the diseased joint, but involves the upper part of the muscle in the same degree as the lower part. The degree attained varies much in different cases. Usually moderate, and sometimes slight, it is occasionally considerable, so that the femur, for instance, may be readily felt when the quadriceps femoris is the seat of the wasting, or the head of the humerus may be distinct, and acromion prominent, if the deltoid is affected. Although the whole length of the affected muscle is always involved, if the muscle is a wide and compound one, some parts may suffer chiefly. Thus in the thigh all parts of the extensor may waste equally, or the rectus or vastus internus may suffer more than the other parts. The wasting increases during two or three weeks, then becomes stationary, and continues as long as the joint disease lasts. When the joint has recovered, the muscles in most cases slowly regain their normal size. Occasionally, especially when the arthritis has lasted a long time, the wasting may continue for months or years after the joint disease is at an end.

The electrical irritability of the atrophied muscles may be normal, but is often slightly lessened, equally to faradism and voltaism. The change is trifling, to be recognised only by comparison with the other side. I have found that this arthritic atrophy is generally accompanied by a distinct, and sometimes a considerable, increase in myotatic irritability. The knee-jerk is excessive, if the thigh-muscles are affected, and a rectus-clonus can sometimes be obtained. A foot-clonus may be elicited when the ankle-joint is affected. Occasionally the increased irritability extends beyond the region of atrophy, so that, for instance, a foot-clonus may be obtained when the knee-joint and thigh-muscles are affected, although there is no wasting below the knee. As an

example of this, which illustrates also the occasional persistence of the atrophy, may be mentioned the case of a young man who jumped over a hoarding and twisted his left leg in doing so. He felt immediately severe pain in the knee, the joint quickly swelled, and became the seat of an acute inflammation which lasted for several weeks, and then slowly subsided. During the inflammation, the thigh wasted. I saw him two years after the onset, and there was still considerable wasting, involving the whole of the extensor of the knee. The minimum circumference of the left thigh above the knee was three quarters of an inch less than that of the right, although there was no difference between the two legs below the knee. There was a slight diminution in faradaic and voltaic irritability in the affected muscles. The knee-jerk was much more considerable on the left than on the right side, and was obtained from above, and there was a well-marked foot-clonus in the left leg, but none in the right. He complained that the leg became tired sooner than the other. A year and a half later the difference between the two thighs was still the same, and the other symptoms were unchanged. In some cases there is slight rigidity of the affected muscle; we have seen that rigidity is often associated with an increase of myotatic irritability. Very rarely some contracture occurs in the opponents of the atrophied muscles, but it is uncertain how far the atrophy contributes to this. Thus a child, aged eight, had an attack of rheumatic fever succeeding chorea; the inflammation in the knee-joints was prolonged, and was accompanied by marked arthritic wasting in the thighs. During the confinement to bed, the flexors of the knees became contracted. There was only a slight diminution in the electrical irritability of the muscles, and no distinct paralysis. Sensory symptoms are as a rule absent. I have occasionally known slight tingling in the skin to be complained of, and in one case this symptom was persistent. It is said that areas of anæsthesia sometimes develop, but it is probable that, in such cases, inflammation has extended to a nerve in the vicinity of the joint.

PATHOLOGY.—We have no facts regarding the morbid changes that occur in man. In animals Valtat found the muscles paler than normal, with simple narrowing of the fibres, but without interstitial changes, and no abnormal appearance could be detected in the nerves or the spinal cord. Thus pathological anatomy throws no light on the mechanism by which the wasting is produced. The only constant condition, in these cases, that can be regarded as the cause of the wasting, is the simple fact of the joint disease. The atrophy seems to be especially rapid when there is much pain in the joint, and Paget has observed similar wasting when there has been neuralgic joint-pain without inflammation. But such pain is not always attended by wasting, and the latter may attend arthritis that is not painful. Disuse will not explain the atrophy; first because its influence in causing wasting is trifling and tardy, and secondly because

other muscles of a limb, equally disused, present no wasting. Nor can the effect be due to any local inflammation of the muscles, passing to them from the inflamed joint. Such a cause is excluded by the fact that the whole length of a muscle suffers equally; the part at a distance from the joint wastes as much as the part near the joint, and moreover, when such wasting occurs in animals, from traumatic arthritis, no signs of inflammation can be found either by simple or microscopical examination. We are, therefore, reduced to the assumption that the wasting is produced, in some way, through the nervous system. "A reflex atrophy, due to the disturbance of some nutritive nervous centre, irritated by the painful state of the sensitive nerve-fibres" is the pathology suggested by Paget. A similar explanation has been given by Vulpian, who surmised that the wasting may be due to alterations in the nutrition of the ganglion cells of the cord. A theory of this character seems the only tenable one. It is, however, an interesting fact that the wasting is accompanied by an increase in the myotatic irritability. We have, in fact, the condition that often results from slight degeneration of the pyramidal tracts, and must be immediately due to the changes in the termination of the pyramidal fibres in the grey matter—increased myotatic irritability, with slight wasting, and merely a slight diminution in the electrical irritability, similar to each current. It is possible, therefore, that this terminal structure participates in the nutritional changes. This view is favoured by two other facts, first, that the increased myotatic irritability may continue for years, and secondly, that a primary joint inflammation has been known, in rare cases, to cause persistent symptoms of impaired function of the pyramidal fibres—presumably the result of changes in the nutrition of their terminations—without muscular wasting (see p. 330). Since the sensory influence must be exerted through the sensory cells of the cord, we can understand the occasional occurrence of such subjective sensations as the tingling above mentioned.

DIAGNOSIS.—The chief difficulty in the diagnosis of the condition arises from ignorance of the occurrence of this form of wasting. Its nature is usually recognised at once, if its existence is known, because the primary character of the arthritis which produces it is sufficiently obvious. In cases in which a joint inflammation results from a primary disease of the nervous system, which also causes wasting of the muscles, a mistake is conceivable, but it is unlikely, because such arthritis results only from lesions of the nervous system that are considerable in degree, and their existence is then sufficiently obtrusive. In some cases of neuritis in the arm, secondary arthritis may occur in the small joints of the hand, with wasting of their muscles, and the ultimate condition may very closely resemble, in aspect, that which we are now considering, but other indications of a serious lesion of the nerves, such as anæsthesia and paralysis, either are or have been present, and indicate the nature of the case.

PROGNOSIS.—When the inflammation of the joint is brief in duration, recovery of the muscles may be anticipated with confidence. In children, even after prolonged joint disease, the muscles usually regain their normal bulk. In adults the wasting often lasts for a long time after the joint is well, and a cautious prognosis should be given, if the arthritis has lasted long. Even if slight wasting is persistent, normal power is usually recovered, but, as the case mentioned above shows, the symptoms sometimes continue for years.

TREATMENT.—The chief treatment is local; electrical stimulation of the muscles, and gentle rubbing. It is doubtful whether drugs have any influence on the condition. As long as the joint is inflamed, treatment rarely causes any increase in the bulk of the muscle. When the arthritis has ended, the muscles usually recover slowly without assistance, but it is probable that the local treatment accelerates the process. The form of electricity is of little consequence; either faradism or voltaism may be employed, but should only be used in sufficient strength to cause gentle contraction of the muscles. Painful stimulation of the sensory nerves should be avoided.

MUSCULAR ATROPHY FROM OVER-USE.

Muscles that are much used sometimes waste. The effect is seen most frequently in the small muscles of the hand, in persons of weakly constitution, who use these muscles unduly. Thus a young lady devoted many hours a day to "illuminating," during several years, and then some muscles of the thenar eminence began to waste, and became considerably atrophied. Such wasting is scarcely ever met with in the larger muscles, but has been observed in the biceps, consecutive to hypertrophy, in Sheffield smiths (Frank-Smith). The electric irritability is gradually lowered in this local wasting, equally to both currents, as it is in progressive muscular atrophy. We do not know whether this wasting from over-use is purely local or whether the related ganglion cells of the spinal cord fail first, and the muscular wasting is secondary to their atrophy. As Gull said many years ago, "It is as reasonable to infer a lesion of the grey matter from overwork as of the muscles."* The wasting often persists when the over-use of the muscles is discontinued. Hypertrophy from over-use does not usually give place to atrophy. These two facts are somewhat in favour of the view that the lesion is primarily of the nerve-cells. The wasting in these cases shows no tendency to extension beyond its original limits. The treatment is, first, the cessation from the excessive exertion of the muscles; secondly, the improvement of the general health, and the administration of nerve tonics, especially of strychnine; and thirdly, the gentle electrical stimulation of the muscles by either faradism or voltaism.

* 'Guy's Hosp. Reports,' 1862, p. 246.

IDIOPATHIC MUSCULAR ATROPHY

(MYOPATHIC ATROPHY; MUSCULAR DYSTROPHY).

Idiopathic atrophy, which is primarily muscular and does not depend on disease of the central nervous system, may be conveniently considered in this place. It does not indeed come within the class of diseases that are the subject of this book, but it is one of those maladies in which clinical resemblance may conveniently be allowed to supersede the rules of pathological classification.

The chief form of idiopathic muscular atrophy is that known as "pseudo-hypertrophic paralysis." Other cases occur which resemble more closely in their aspect the spinal form, and differ in some particulars from the pseudo-hypertrophic disease, with which, however, they have some characters in common.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS (MUSCULAR PSEUDO-HYPERTROPHY; LIPOMATOUS MUSCULAR ATROPHY, &c.).

The affection thus designated is a disease which chiefly manifests itself during the later developmental period of childhood, and clearly depends on a morbid developmental tendency which is often present in several members of the same family. It is thus a congenital disease in its origin and nature, although not congenital in its manifestation. It is characterised by a progressive change in the size, and diminution in the power, of many muscles. They may be larger than normal, or smaller, or may be at first large and then small, and they may be harder than normal. The alterations in size and consistence depend on an abnormal growth of interstitial connective tissue, in which fat is often deposited, and before which the muscular fibres waste. The progressive weakness cripples the patient, and interferes with breathing power, until chronic lung disease or some intercurrent malady ends life.

Isolated cases, which can now be recognised as examples of this disease, were recorded in England in 1830 (Sir Charles Bell) and in 1847 (Partridge) and in Italy in 1838, but the first series of cases were described by Meryon in 1852, and Oppenheim in 1855. These observers failed, however, to distinguish the disease from the common form of progressive muscular atrophy, partly because enlargement of muscles was not so obtrusive a feature of these cases as it was in some others which soon afterwards came under the keen eye of Duchenne. This acute observer was then exploring the field of muscular paralysis with the aid of "localised electrification." He recognised at once the novel features of the disease, and published, in 1861, an admirable account of it, giving to it the name of "pseudo-hypertrophic muscular

paralysis," which, in spite of its clumsiness, expressed the salient features of the malady sufficiently well to have since held its own against many other designations that have been suggested by more exact knowledge of the pathological changes.

ETIOLOGY.—Our knowledge of the causes of the disease is limited to a few general facts. Males furnish the majority of the cases, but the actual degree of preponderance is not yet certain. Published cases show a proportion of seven to one, but of forty-three cases that have come, directly or indirectly, under my own notice, thirty-three were males and ten females. This discrepancy is perhaps due to the fact that the slighter liability of females influences the character of the disease as well as its occurrence. In them it is slighter in degree, later in development, and less frequently causes death. Hence the disease is more likely to escape notice in females, and cases in which it is recognised are less likely to be published, because, when a disease has become familiar, the cases recorded are chiefly those, as we say, "completed by an autopsy." Hence it is probable that a proportion of four males to one female is near the truth.

The disease occurs rather less commonly in isolated cases than in family groups. The number in a family has varied from two to eight. As many as eight brothers suffered and died in the family described by Meryon, while all the daughters escaped. In a family known to me, four sons have suffered and none of the daughters; in another instance two daughters are affected and no sons. Thus there may be a tendency in a family to the affection of one sex, and not the other, but, on the other hand, it is also common for both sexes to suffer in the same family. In many instances in which many members of one generation are affected, no antecedent cases can be traced in the family; the malady, while congenital, is not hereditary. In other families antecedent cases can be traced, and these are invariably on the mother's side. The disease is thus transmitted by women who are not themselves its subjects. In a case in which four brothers suffered, the mother's brother and sister were likewise affected. Again, a brother and sister were diseased, one daughter of a second sister, and three daughters of a third sister. In another instance a boy suffered, and his sister, unaffected, had two sons diseased and a daughter free, of whose children two sons were the subjects of the malady. Thus the congenital tendency is exclusively due to the maternal element in the embryo. This is also shown by another fact, that the children of the same woman, by different husbands, have been affected. Indirect hereditary tendencies, such as are indicated by the occurrence of diseases of the nervous system, can be traced so rarely that it is doubtful if they have any influence, and it does not appear that the occurrence of the malady is due, in any degree, to mere consanguinity of parents, to their age, or their intemperance.

The disease always manifests itself during the period of development,

sometimes in the early stage of growth, at the close of infancy, often only during mid-childhood, rarely not until growth is nearly ended. In a third of the cases, the first symptoms are noted when the child first attempts to walk, which is usually a little later than in healthy children. In about another third the child seems well until it is four, five, or six years old, and then impairment of power attracts attention. In three quarters of the cases, the disease manifests itself before the tenth year. Very rarely the patient is conscious of no symptoms until after puberty, and he has reached the age of eighteen or twenty. I have seen four instances of this late apparent onset, but in each inquiry has elicited a history of enlargement of muscles long before their power became impaired. One patient, for instance, in whom weakness was only noticed when she was twenty, had been often "chaffed," when a young girl at school, on account of her "tea-kettle calves." It is therefore probable that, in such late cases, it is rather the influence of the disease on the muscular fibres, than its development, which is delayed. Neither social state nor general constitutional condition seems to influence the occurrence of the disease, but its manifestation has sometimes been apparently accelerated by influences that disturb the general health; it has been first noticed, for instance, during convalescence from some general illness.

SYMPTOMS.—Impairment of power usually attracts attention before any change is observed in the size of the muscles, or if these are noticed to be large, it is with feelings of parental pride rather than with suspicion. The children, who usually walk late, often also walk clumsily, fall with ease and rise with difficulty. The act of going upstairs is especially difficult to them; the child has to take hold of the banisters and pull himself up.

The muscles may at first present nothing unusual; if the child is fat, as is frequently the case, neither an increase nor a diminution in size can be readily observed. At the age of five or six years an unnatural enlargement of certain muscles is usually conspicuous, especially when there is a contrast between these and other muscles which are small. If enlargement is almost universal it is usually great and conspicuous. The enlargement of muscles, although for a time it may increase, ultimately lessens, earlier in some muscles than in others, and it may give place to an actual wasting.

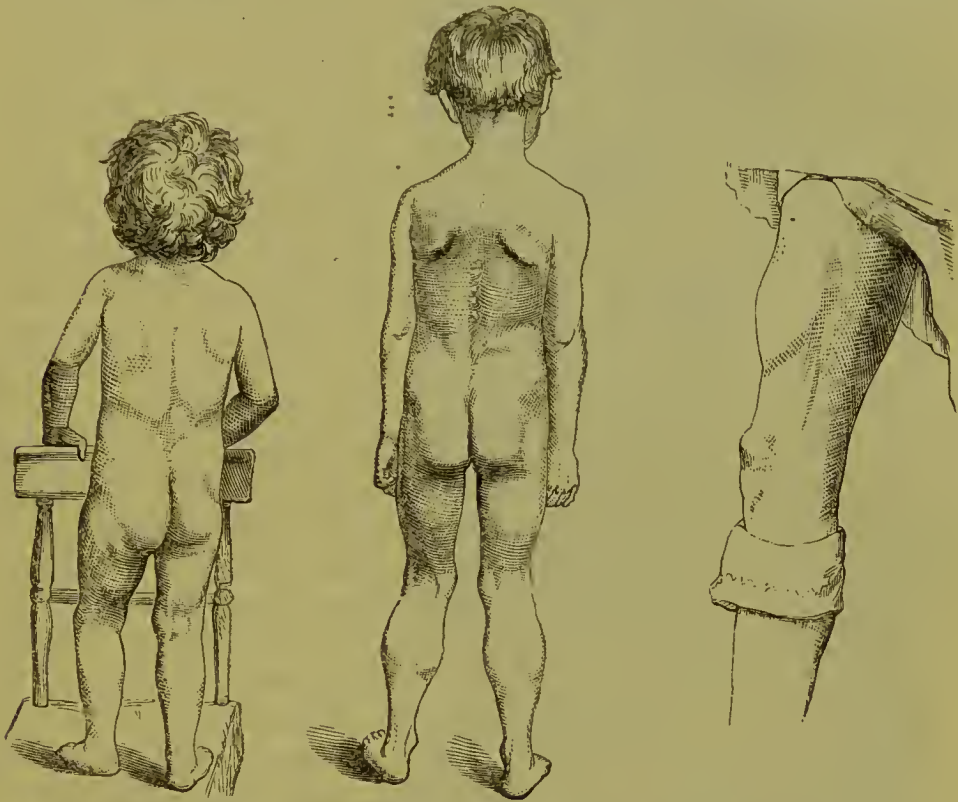
Among muscles that are most frequently large those of the calf take the first place. They sometimes attain a remarkable size. I have measured a calf $14\frac{1}{2}$ inches in circumference in a boy of twelve. The muscles in front of the lower leg are less frequently enlarged, but sometimes project beyond the edge of the tibia. The extensors of the knee are often big; occasionally the rectus or vastus internus is alone increased in size (the rectus, in Fig. 139), and the other parts may be normal or small; less frequently all parts are small. The flexors of the knee commonly escape, the glutei are frequently conspicuously large, and

so also are the lumbar muscles. The condition of those of the shoulder is peculiar and important. Of all the muscles of the body, next to those of the calf, no one is enlarged more frequently or in greater relative

FIG. 137.

FIG. 138.

FIG. 139.



FIGS. 137 and 138.—Two brothers, aged four and seven, suffering from pseudo-hypertrophic paralysis.

FIG. 139.—Partial enlargement of rectus, the vasti being small.

degree than the infraspinatus. It often stands out so conspicuously that its limit is apt to be mistaken for the edge of the scapula (Figs. 140 and 141). The supraspinatus is sometimes also prominent, but its condition is usually concealed by the trapezius, which is little involved. The deltoid is also frequently large; the serratus rarely. The pectoralis is never enlarged, but on the other hand, its lower half is wasted in a large proportion of the cases, and with this the latissimus dorsi, which has the same action in depressing the raised arm (see p. 26). The teres major may share the wasting of the latissimus. In many cases no trace can be found of the latissimus and lower part of the pectoralis, and it even seems as if, in some cases, these muscles may be congenitally absent. In consequence of the loss of the latissimus, the posterior axillary fold is almost absent, and the aspect of the axilla is strikingly abnormal, especially when, as is often the case, the defect contrasts with the conspicuous prominence of the infraspinatus (Figs. 140 and 141).

The other muscles of the arm suffer in diminishing degree and frequency from above downwards. The triceps and biceps are sometimes

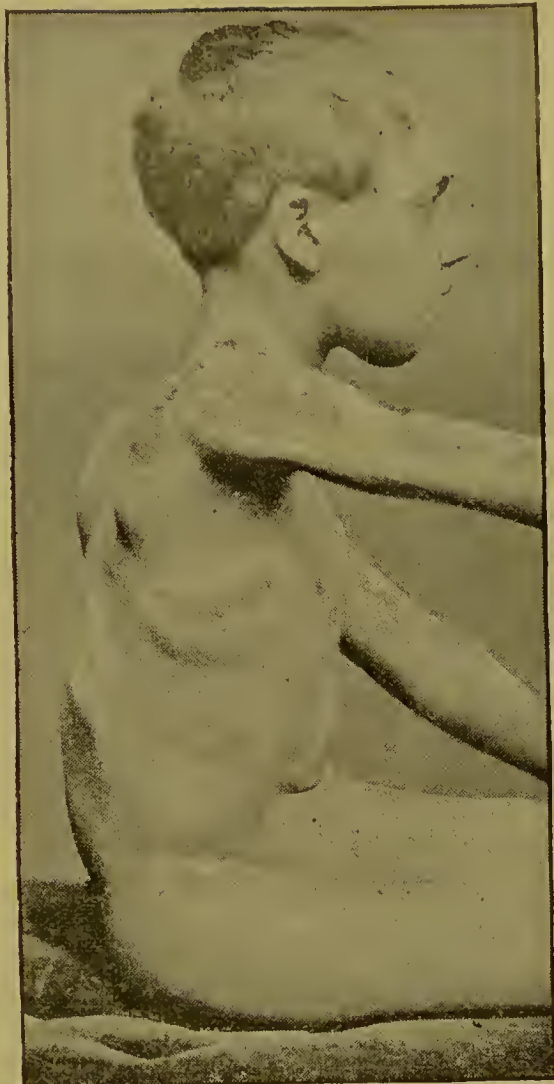
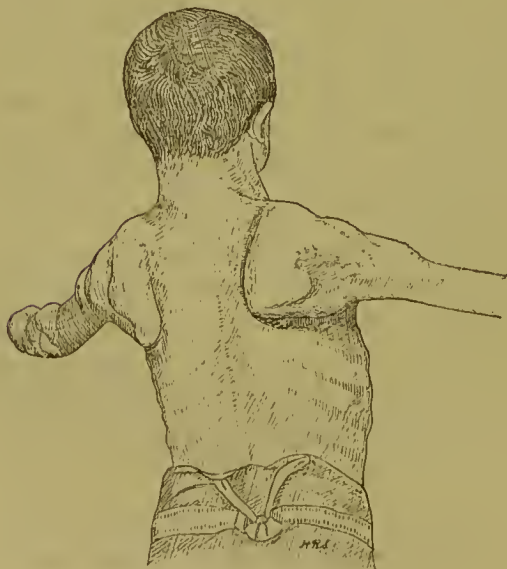


FIG. 140.—Pseudo-hypertrophic paralysis. Absence of latissimus dorsi, enlargement of infraspinatus. (From a photograph.)



enlarged, the former more frequently than the latter, but occasionally only in one part. Both these muscles are sometimes wasted. The forearm muscles suffer in only a small minority of the cases, and the intrinsic muscles of the hand usually escape altogether.

The muscles of the neck are rarely affected, but I have noted, in a few cases, wasting of the clavicular part of the sterno-mastoid. Those of the face do not suffer, but in the patient shown in Fig. 144 there was considerable enlargement of the masseters. The tongue has been increased in size in a few instances. The other muscles supplied by the cranial nerves always escape.

The diseased muscles are weak, but the impairment of power is to some extent irrespective of the change in size. The muscles that are abnormally small are generally weaker than those that are abnormally large; and in the latter the weakness increases with the wasting. In the legs the greatest weakness is in muscles that are inaccessible to observation,—the flexors of the hips; next in order of weakness come the extensors of the knee, and the

FIG. 141.—Wasting of latissimus dorsi and serratus; enlargement of infraspinatus, supraspinatus, and deltoid; atrophy of biceps and triceps. (By Dr. H. R. Spencer, from a photograph.)

extensors of the hip. The muscles below the knee usually retain considerable power for a long time, and the extensors of the ankle fail before the flexors. In the upper limbs the depressors of the arm are usually alone weakened during the early period of the disease, but subsequently the shoulder-muscles suffer, then the triceps and the biceps, while the muscles that move the hand commonly retain good power to the last.

The distribution of weakness in the legs causes certain peculiar defects of movement which are very characteristic, and some are even all but pathognomonic of the disease. The difficulty in going upstairs is especially due to the weakness of the extensors of the knee and hip. The defect of the extensors of the hip causes the gait to have a peculiar oscillating character, in which the body is so inclined as to bring the centre of gravity over each foot, on which the patient successively throws his weight, because the weak gluteus medius cannot counteract the inclination towards the leg that is off the ground, unless the balance is exact. The greatest defect, however, is in the power of rising from the floor, and the most characteristic peculiarity is the mode in which this is achieved, if still possible, and no objects are near by which the patient can aid himself. He commonly has not sufficient power to extend the



FIG. 142.—Mode of obtaining extension of hips in pseudo-hypertrophic paralysis. F, fulcrum of the lever formed by the femur. P, mean position at which the power is applied by contraction of the quadriceps femoris. W, position of weight in the ordinary mode of rising. w, the place to which part is transferred by putting hands on knees.

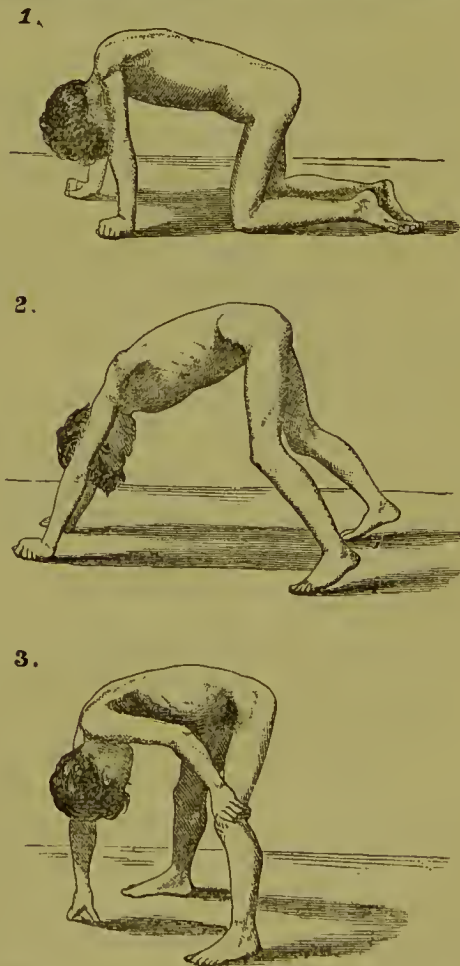


FIG. 143.—Mode of rising from the ground in pseudo-hypertrophic paralysis.

knees when the weight of the trunk is on the upper extremity of the femur, which is then a lever in which the power, applied between the fulcrum and the weight, acts at least advantage. He therefore places his hands on his knees, as in Fig. 142; and his arms thus bring much of the weight of the upper part of the trunk—on the femur close to the fulcrum, between this and the power, which can then act at greater advantage. Moreover, the mere weight of the head, which is in front of the arms, tends to aid the extension, and indeed may effect the extension of the knee without the aid of the extensor muscles, as anyone may ascertain by observing the mobility of the patella in this attitude. When the knees are extended, the power of the extensors of the hip may be sufficient to raise the body into the upright position, or the patient may aid them by an upward push with the hand as he takes it off. If, however, these extensors are weak, the hands are often moved higher and higher up the thighs, grasping alternately, and thus pushing up the trunk. To get thus the requisite support, the knees must not be quite extended, and if their extensors have no power, the device cannot be employed, and the patient is altogether unable to rise. In many cases, especially when extension of the hip is easy, the patient achieves the extension of the knees in another way; he puts the hands on the ground, stretches out the legs behind him far apart, and then, the chief weight of the trunk resting on the hands, by keeping the toes on the ground and pushing the body backwards, he manages to get the knees extended, until the trunk is supported by the hands and feet, all placed as widely apart as possible (Fig. 143, 2). Next the hands are moved alternately along the ground backwards, so as to bring a larger portion of the weight of the trunk over the legs. Then one hand is placed upon the knee (Fig. 143, 3) and a push with this, and with the other hand on the ground, is sufficient to enable the extensors of the hip to bring the trunk into the upright position.

The shortening and contraction of certain muscles lead to another group of symptoms,—distortions due to permanent alteration in the position of joints. Some of these are produced, as are distortions in other forms of muscular weakness, by shortening of the less affected opponents of the weaker muscles. Thus the knee-joints become fixed by the contraction of the flexors, and the elbow by the contraction of the biceps when the triceps has lost all power. These contractions only occur late, and are usually facilitated by the habitual flexion of the knee- and elbow-joints. But the deformity at the ankle-joint, which results from contraction of the calf-muscles, commences at a much earlier period, before there is any disproportionate weakness in the opponents of these muscles, and with little assistance from posture. It coincides commonly with the diminution in bulk of the muscle, and is the result of the shrinking that occurs in the length as well as in the breadth of the muscle,—a primary contraction. As a consequence of it the patient cannot get the heels well upon the ground, and the foot

cannot be flexed passively beyond a right angle. The gradual increase of the contraction results in considerable "talipes equinus." In consequence of the muscular weakness which coincides, the patient is able to walk but little, and the loss of the extension involved in the act of walking permits a rapid increase in the unopposed contraction. The feet, as Fig. 144 shows, soon assume a posture of extreme extension, the dorsum being in a line with the front of the leg, or forming with it a convex curve. A subluxation of the ankle-joint takes place, and the articular surface of the astragalus, its anterior extremity, and that of the os calcis, form three prominences under the skin. When this reversal of the ankle occurs, the tibialis anticus can no longer act as a flexor.



FIG. 144.—Late stage of pseudo-hypertrophic paralysis: a boy fourteen years old, with muscular contraction and wasting and lateral curvature of the spine.

Another deformity, which is due chiefly to muscular weakness, is curvature of the spine. An antero-posterior curve, with the concavity backwards, is an early symptom of the disease, and it may become extreme, the upper part of the trunk being carried so far back that a vertical line from the scapula falls an inch or more behind the sacrum. It is due, not to the weakness of the trunk-muscles, but to that of the extensors of the hip, in consequence of which the



FIG. 145.—Lad, aged fifteen; late stage; wasting of thighs; inability to sit upright in consequence of the weakness of the spinal muscles.

pelvis is inclined forwards, carrying with it the lower lumbar vertebræ and the abdomen; hence the upper part of the trunk has to be held far

back to keep the centre of gravity of the body over the feet. The proof of this mechanism is that when the patient sits, and the pelvis is supported on the ischial tuberosities, the lordosis disappears. It is, indeed, replaced by an opposite curve, in which the back becomes convex, clearly due to the weakness of its extensor muscles. This curve may become very great, as in the case shown in Fig. 145. This weakness of the spinal muscles also permits the occurrence of lateral curvature (Fig. 144), influenced, in its direction, by the habitual posture and the preponderance of weakness on one side or the other.

The electric irritability of the muscles is not usually altered until distinct weakness is developed. Then, especially after wasting sets in, the irritability is lowered, alike to faradism and voltaism. There is never any trace of degenerative reaction.

The knee-jerk may be at first normal, but as the extensors of the knee become feeble it is always lessened and gradually disappears. It is never excessive, and in all advanced cases it is lost. Sensation is unaffected, and so also are the sphincters in the vast majority of cases. Very rarely there has been, towards the end, a slight difficulty in the retention or expulsion of urine, to be regarded, perhaps, rather as a complication than as an effect of the disease. All other functions of the nervous system are commonly normal. The sympathetic functions are undisturbed, and in most cases the mental development of the subjects of this disease is rather beyond that of other children of the same age, doubtless on account of the indirect influence of a malady which withdraws them from active amusements.

The rate of progress of the disease and its duration vary much. After some years, often between ten and fourteen, the power of standing becomes lost in consequence of the increasing weakness and the contraction of the calf-muscles. The latter is chiefly influential, and if the tendons are cut, the ability to stand may be restored and may remain for some years more. When the patient ceases to walk, the muscular disease makes more rapid progress, deformities become greater, and lateral curvature of the spine intensifies the effect of the muscular weakness in limiting the respiratory movements. The muscular wasting may then become great, especially about the shoulders and thorax, but the patient may live on, helpless, except in the hands, for several years. Death is often due to some intercurrent malady, an acute specific disease, acute pneumonia or bronchitis. In other cases death is the result of chronic lung disease, a form of pneumonic phthisis or broncho-pneumonia, which develops gradually, with little febrile disturbance, apparently in consequence of the influence on the lungs of the lessened respiration. There is never sufficient impairment of the respiratory muscles to cause death directly. Such pulmonary disease generally ends life some time between twelve and twenty. The duration of the stage of helplessness depends very much on the care which the patient can obtain.

In the cases in which muscular power remains good until after

puberty, the progress of the disease is generally slow. The patient may reach the age of thirty before power is much impaired. It is possible that, in some cases, the disease never attains a considerable degree. More frequently, however, after slight symptoms have lasted for some years, a rapid increase occurs, and very few patients reach the age of forty. The course of the disease is slower in girls than in boys, and females furnish a relatively large proportion, perhaps one half, of the late cases.

Varieties.—The chief varieties of the disease depend on the age at which it commences, and on the condition of the muscles, whether they are large or small. In rare cases a single muscle may be large, and the rest small, as in Fig. 146, in which only the vasti were increased in size. Or everywhere and from the first the muscles are smaller than normal, and they progressively waste. Such cases are not uncommon; in the first group of cases described (by Meryon), enlargement of muscles was inconspicuous. Many cases in which all muscles are small belong properly to the form considered in the next section. The cases described by Meryon are generally regarded as examples of pseudo-hypertrophic paralysis, but they present many points of resemblance to the "simple atrophy" described further on.

Complications.—Congenital mental weakness, due apparently to defective development of the brain, sometimes complicates pseudo-hypertrophic paralysis. In rare cases there have been indications of some other morbid condition of the central nervous system, such as epilepsy. It is uncertain in what light the slight occasional affection of the bladder is to be regarded, whether as an invasion of the vesical muscles or as a central complication. Vigoroux has recorded a case in which the symptoms of pseudo-hypertrophic paralysis were combined with the peculiar rigidity of Thomsen's disease. Of course the subjects of the disease are liable, like other children, to various diseases of the nervous system, such, for instance, as chorea, which brought one well-marked case under my notice, but these are merely accidental complications.

PATHOLOGICAL ANATOMY.—It is rare, at the time of death, for any muscles to be actually larger than natural, and most of those that are affected are usually below the normal size. They are pale and yellowish in colour, and often, to the naked eye, resemble perfectly masses of adipose tissue. The resemblance is not merely one of aspect. As seen under the microscope, it may be difficult for the observer to realise that he is not looking at a fatty tumour. Nothing may be at first visible but fat cells, precisely like those of adipose tissue. Among

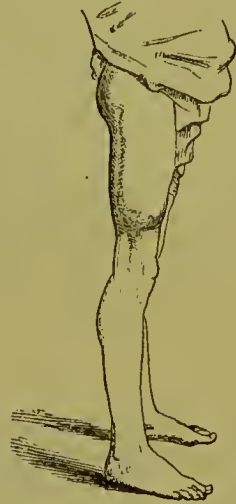


FIG. 146.—Enlargement of the vasti and not of the rectus. In this case all other muscles were below normal size.

the cells, however, are tracts of nucleated fibrous tissue, and a closer examination of these shows that the tracts contain also muscular fibres

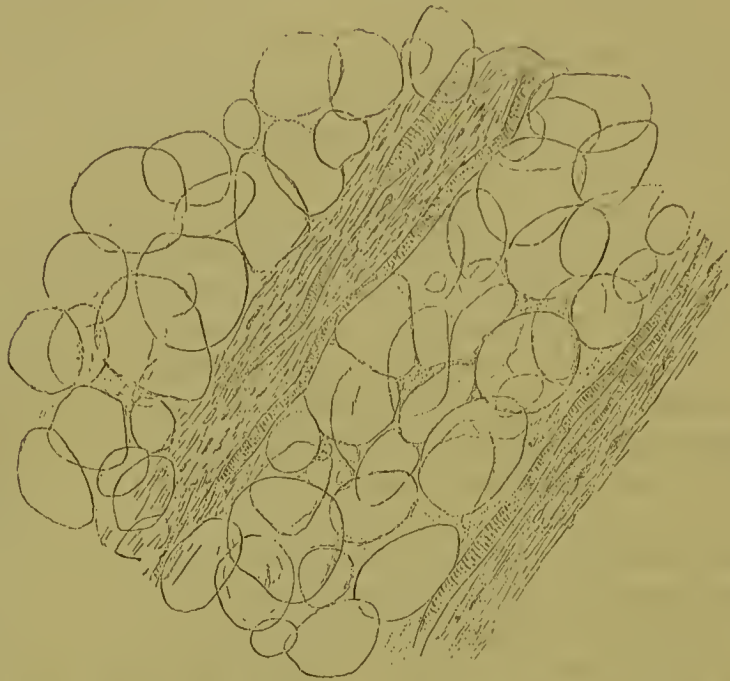


FIG. 147.—Gastrocnemius muscle; muscular fibres, irregularly narrowed and in part degenerated, lie among tracts of nucleated fibrous tissue, separated by adipose tissue.

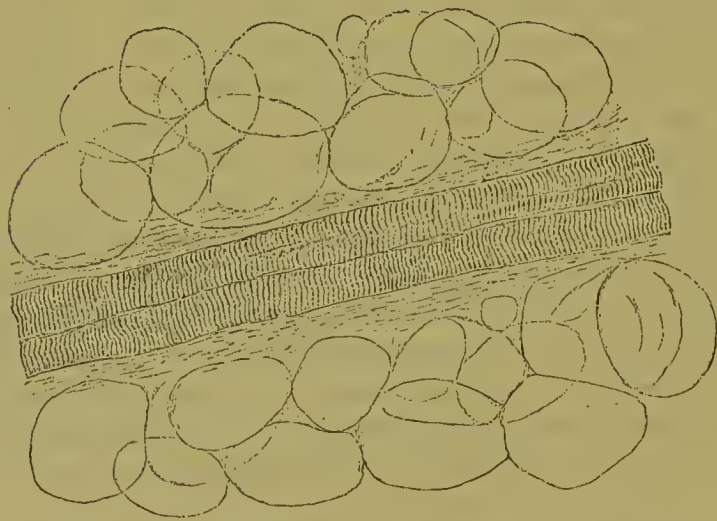


FIG. 148.—Gastrocnemius muscle; two nearly normal muscular fibres, accompanied by fibrous tissue, surrounded by fat cells.

(Fig. 147), most of them much narrower than normal. They are also irregular in width; a broad fibre, for instance (as in the figure), suddenly becoming narrow.

The fibres for the most part preserve their transverse striation, but where they are narrowest this may have in part disappeared, either by

granular degeneration, or, more commonly, by a simple fading of the striæ. In the narrowed fibres the striæ are sometimes farther apart than normal. In other parts broad fibres may be seen, normal or nearly normal in aspect (Fig. 148) coursing among the fat cells, and accompanied by a smaller amount of fibrous tissue. Large fibres occasionally present fatty degeneration, or a longitudinal striation, but these are rare. Some empty sarcolemma sheaths may be seen where the narrowing of the fibres is greatest. In muscles that still preserve some red tint, the amount of fat is less, and there is often a relatively larger amount of interstitial fibrous tissue.

In some of these the interstitial tissue may be almost entirely fibrous, a few fat cells only being visible here and there. In such muscles it is common to find the fibres more damaged than in those in which the growth is partly fatty. It is chiefly in the latter that many normal fibres are seen. No muscular fibres larger than normal have been found after death, but such fibres have been seen in fragments removed during life by excision or by a "harpoon-trocar;" it is probable that the increased size was due to a vital contraction under the mechanical stimulus involved in the extraction. (See note, p. 410).

The motor nerves to the muscles have not in any case been found diseased. The condition of the sensory muscle-nerves (which terminate in the interstitial tissue in which the primary morbid process occurs) has not been ascertained in any instance. In several cases in which the spinal cord has been examined it has been perfectly normal. In a few there have been slight and irregular degenerative changes. They have not been so marked in any other case as in one examined by Lockhart Clarke and myself.* In this case the cervical and dorsal regions were normal, with the exception of here and there slight accumulations, at the bottom of the fissures, of products of degeneration, probably derived from the perivascular erosion common at all ages. At the last dorsal segment, however, there was an area of granular disintegration in the intermediate grey substance on each side, in front of the posterior vesicular tract. This part was unduly translucent for half a centimetre in vertical extent, and in the middle of this area the disintegration had produced an actual cavity across which the fibres for the cerebellar tract ran unchanged.

PATHOLOGY.—The common integrity of the anterior grey matter of the cord, and especially of the motor nerve-cells, seems conclusive evidence that the disease of the muscles is not due to a primary lesion of the spinal cord. Pseudo-hypertrophic paralysis is not therefore, as was at first thought, merely a form of spinal muscular atrophy with a special muscular change. The significance of the pathological anatomy is that the malady is a primary disease of the muscles, consisting in an overgrowth of the interstitial connective tissue, in which fat may

* 'Med.-Chir. Trans.,' vol. lvii, p. 217.

or may not be deposited, and that the wasting of the muscular fibres is secondary to the interstitial growth. The significance of the conditions under which the disease occurs is that the disease is congenital, the result of a perverted tendency of growth, inherent in the embryo, and derived from the germ from which the embryo proceeds. In this connection it is instructive to note that there is one form of congenital tumour the structure of which is almost exactly the same as that of the muscles in pseudo-hypertrophic paralysis. Fig. 149 might be a



FIG. 149.—Section of a myo-lipoma which was attached to the spinal cord of a man suffering from locomotor ataxy, for comparison with Figs. 147 and 148.

fragment of a muscle in this disease, but it is a section of a myo-lipoma attached to the conus medullaris of the spinal cord of a patient whose muscles were healthy. (The tumour is shown also in Fig. 155.) Such a tumour, in such a situation, must have been of congenital origin, due to the misplacement of some of the embryonal elements from which muscular tissue is developed, and it shows that from such germ tissue the structural condition found in pseudo-hypertrophic paralysis may arise.

The essential lesion is the growth of connective tissue, by which the fibres are damaged, whether fatty tissue is formed or not. Indeed, the fibres seem to suffer more when there is only fibrous tissue than when there is the fatty deposit. It is chiefly the fat that causes the enlargement of the muscles. There seems to be a tendency to the formation of fat in the early stage of the disease, and to its removal in the later stage, since in the former the muscles may often be observed to increase in size, and in the latter they become smaller, partly from some removal of fat and partly from the increasing atrophy of the muscular fibres. It is then that the greatest impairment of power occurs. In the cases in which the muscles are small from the first the tendency to the deposit of fat seems slight, and the condition is an almost pure muscular sclerosis. The late shortening which occurs

seems to be due to the contraction of the interstitial fibrous tissue, and it often coincides with the shrinkage from the removal of some of the fat and the atrophy of the muscular fibres.

If it be true that the latissimus dorsi and lower half of the pectoralis are sometimes congenitally absent in pseudo-hypertrophic paralysis, the fact is quite consistent with the explanation of the nature of the disease given above. These two muscles stand perhaps lowest, in functional importance, among the muscles of the body, being used chiefly for the rare depression of the arm against a resistance (see p. 26). It is readily intelligible that a defect in the embryonal tissue of the muscular system should be quantitative as well as qualitative.

The loss of the knee-jerk is sufficiently explained by the muscular lesion. According to the theory that the irritability on which the jerk depends is due to a muscle-reflex action, the loss is readily intelligible, since the afferent impulse is due to the stimulation, by tension, of the sensory muscle-nerves, and these end (or begin) in the interstitial tissue which is the seat of the primary morbid process in this disease. But the wasting of the muscular fibres must also be capable of abolishing the knee-jerk when the atrophy reaches a considerable degree, and it is also possible that the motor nerve-endings share the structural damage. On any theory of the nature of the knee-jerk, this will explain its loss. It is only when the muscular changes have attained a considerable degree that the loss occurs. The fact therefore does not, in itself, suggest any lesion of the spinal cord.

DIAGNOSIS.—The diagnosis of the disease is usually easy, if its characters are known. The peculiarity of gait and the mode of rising from the floor, the age of the patient, and the progressive character of the impairment, are in themselves sufficient to suggest the disease, and examination reveals enlargement and often contraction of the calf-muscles, and a change in the size of others, which confirm the diagnosis. The mode of rising is not absolutely pathognomonic, because it is present in other diseases which cause a gradual weakening of the extensors of the knee, but such gradual paralysis is exceedingly rare in early life, from any other cause. Hence the peculiarity is of great diagnostic value, greater even than might be anticipated.

Of the condition of the muscles, that which is most characteristic is the combination of enlargement of the infraspinatus combined with a wasting of the latissimus and lower part of the pectoralis. I pointed out some years ago* that this condition, when it exists, is of very high diagnostic importance, and subsequent observations have fully confirmed the opinion. The condition is, moreover, rarely absent. The cases in which the latissimus is not wasted are often untypical, and the diagnosis frequently cannot be made with absolute certainty. Next in importance is the enlargement of the calf-muscles, especially

* 'Pseudo-hypertrophic Muscular Paralysis,' London, 1879.

in combination with contracture that cannot be overcome. A distinct enlargement of part only of the extensor of the knee is also of very great diagnostic significance. It is important, however, to remember that enlargement of muscles is not an invariable symptom of the disease. They may be gravely diseased, and yet of normal size, or from the first smaller than normal. In such cases they are often hard, and the distribution of the atrophy is the same as that of the double change in typical cases. But some cases of this character properly belong to the form described in the next section.

The disease which is most frequently mistaken for pseudo-hypertrophic paralysis is the so-called "congenital spastic paraplegia," described at a preceding page. Both diseases affect children; in both there are weakness of the legs and contraction of the calf-muscles, and in both the muscles are frequently large. The chief points in the diagnosis have been already mentioned. The most important are the preservation and excess of the knee-jerk in spastic paraplegia, the tendency to spasm of the legs, the facts that the contracture is active, and can be overcome, that the patient does not rise from the ground, in the way peculiar to pseudo-hypertrophic paralysis, and the opposite tendency of the two diseases. Between spinal muscular atrophy and this disease a difficulty in diagnosis can arise only in untypical cases in which no muscles are large. The age of the subjects is different; the spinal form involves the hand-muscles in most cases, and rarely affects the legs more than the arms, while in many cases the legs are the seat of spasm.

The form of idiopathic muscular atrophy described in the next section is allied to pseudo-hypertrophic paralysis, and if a diagnostic difficulty arises it will chiefly be as to the category in which a case should be placed in which there is no conspicuous muscular enlargement. The most important distinction is the freedom of the calf-muscles from enlargement in the atrophic form. The face is never affected in pseudo-hypertrophy as it is sometimes in idiopathic atrophy. If more than one member of a family is affected, some of the sufferers will usually present characteristic symptoms if the malady is of the pseudo-hypertrophic type, for it is remarkable with what constancy the two forms remain distinct in the various families (see p. 403). If an excised fragment presents no increase of interstitial tissue, pseudo-hypertrophy is excluded, but the presence of some fibrous tissue between the muscular fibres does not prove that the case is of the pseudo-hypertrophic type, since some muscles may present this change in a case of the atrophic form.

PROGNOSIS.—In any case of pseudo-hypertrophic paralysis the prognosis is most grave. It is almost certain that each year will bring increasing disability, and that the patient will not reach adult life. It is only when the disease develops late, and the symptoms do not become considerable until after twenty years of age, that there is a

possibility that the disease may not attain its ultimate degree, but even in such cases this hope is seldom realised. In any case, and at any age, it is unlikely that the patient will live more than seven years after the power of standing is lost.

TREATMENT.—Pseudo-hypertrophic paralysis is one of those diseases against the essential elements of which the physician is powerless, and will probably remain so. So far as our present knowledge goes, a developmental tendency to tissue growth is beyond control. It is not surprising, therefore, that, of the many drugs which have been given in this disease, not one has been found to exert a distinct influence. Arsenic and phosphorus have been thought by some to retard the progress of the weakness, but in no case has permanent arrest been obtained. In a malady of which the course is so prolonged, and in which the process of failure is so gradual, it is easy to fancy that arrest has taken place when the course of the disease is not actually retarded. The stimulation of the muscles by electricity has been employed and advocated, but, however sedulously employed, I have never seen distinct effect from the use of either faradism or voltaism. Indeed, we have no facts whatever to justify the expectation that any form of electricity, that could be applied to the muscles, would influence the interstitial growth of fibrous tissue, or that any electrical stimulation of the fibres can save them from the destructive influence of the compression they endure. It must be remembered, moreover, that electricity is a very feeble agent in stimulating muscular fibres to growth, compared with the physiological stimulus of voluntary effort.

Muscular exercise does seem to have some influence in retarding the failure of power. It may perhaps cause some growth, or increased capacity for contraction, in the muscular fibres that have not yet suffered. It is possible also that it may, to some extent, divert the trophic energy from the interstitial tissue, since cessation of muscular exercise is certainly followed by quicker failure of strength. Hence it is desirable that the patient should carry out carefully planned gymnastic exercises, so arranged as to call into action the muscles that most need help. These, thoroughly persevered in, have seemed, more than any other means, to retard the disease. But they have not in any case arrested it. Rubbing and massage are useful, combined with passive movements, in lessening the tendency to muscular contraction and consequent deformities.

The influence of muscular exercise renders it very important to keep the patient on his legs as long as possible. The ability to stand and walk is generally lost, through the contraction of the calf-muscles, some time before the muscular weakness would take the patient off his feet. In such cases, tenotomy may restore the power of walking for some years, and when contracture returns, its removal has, a second time, set the patient on his feet again. The operation is thus distinctly beneficial. It should be performed without delay as soon as the

actual need for it arises, and division of the tendon is far better than the removal of the deformity by splints, which requires a certain time during which additional structural damage will occur.

During the later stages of the disease great care is required to preserve the patient from catarrh, which helps to excite the pulmonary mischief that so often ends life. Similar care is also needed during any intercurrent malady which the patient may contract.

SIMPLE IDIOPATHIC MUSCULAR ATROPHY.

The name "simple idiopathic muscular atrophy," is perhaps the most convenient term under which to include those forms of widespread muscular wasting, which differ in their characters from the pseudo-hypertrophic disease, and which, according to present evidence, are not central in nature. The affection is a rare one, and the cases present wide differences in character and course. But they possess certain common features by which the disease can be identified with considerable confidence. Of these the most important is the remarkable tendency of the affection to occur in many members of the same family.*

The separation from the pseudo-hypertrophic form is not an absolute one. Intermediate cases are met with, usually isolated, and it may be difficult to decide in which category they should be placed. An obvious distinction is the presence or absence of enlargement of muscles, but we have seen that this is a very inconstant feature of the pseudo-hypertrophic form. Nevertheless there are other differences which will be considered in the account of the symptoms, and it is remarkable with what constancy the two forms keep separate. In those families in which simple muscular atrophy occurs, however numerous the cases, however different the distribution of the disease, and however various the ages at which it begins, no cases present the distinctive characters of pseudo-hypertrophic paralysis.† It may reasonably be anticipated that there is some absolute pathological difference between the two diseases, underlying the difference in their clinical characters and their separate incidence. The anticipation may seem to be verified by the fact that in some cases of simple atrophy there has been no increase of interstitial tissue, but merely a primary wasting of the muscular fibres. But in some other cases there has been such interstitial overgrowth in some muscles, and this when not one of many cases in the family resembled pseudo-hypertrophic paralysis. Hence it is doubtful whether the presence or absence of an increase in the interstitial tissue

* The most important writings on the subject are those of Duchenne in 'Électrisation Localisée' (p. 60 of Poore's translation, published by the New Sydenham Society); Barsickow, 'Inaug. Dissert.,' Halle, 1872; Leyden, 'Klin. d. Rückenm. Krank.,' Bd. ii, p. 525; Möbius, "Hered. Nervenk.," Volkmann's 'Klin. Vorträge,' No. 171; Erb, 'Deut. Archiv f. klin. Med.,' Bd. 34, 1884, and 'Neurol. Centralbl.,' July 1st, 1886; Landouzy and Déjérine, 'Revue de Méd.,' 1885, pp. 81 and 251.

† This is an important fact. We must not actually unite in our classification forms of disease which are habitually separate in occurrence.

can be made a ground of separation, although it is certain that, in some cases of simple atrophy, there is no such increase, and on the other hand that it is invariable in pseudo-hypertrophy.

Among the symptomatic varieties presented by idiopathic muscular atrophy it is easy to distinguish certain forms, especially in regard to the part in which the wasting begins. But if we regard these as distinct types, and attempt to test the validity of the types by ascertaining how far they are associated with differences in the conditions under which the disease occurs, we discover at once that the symptomatic differences do not furnish grounds for the distinction of definite varieties. The types between which there is the most marked difference may all occur in the same family. For instance the atrophy sometimes commences in the face, and Duchenne described this commencement as a characteristic of one variety of muscular atrophy. But in a family in which the atrophy commenced in the face in most cases, it commenced elsewhere in one or two members, and conversely the face has suffered first in one member of a family although the disease began elsewhere in all the others, and the face escaped altogether in some. Thus the test of association invalidates our subdivision of the cases of simple atrophy, as distinctly as it serves us in the separation of simple atrophy from the pseudo-hypertrophic form.

CAUSES.—The ultimate cause in all cases, and in most the only cause which can be traced, is a congenital tendency, often clearly inherited. The proof of this tendency is the occurrence of many cases in the same family, and the evidence of its inheritance is the distribution of the cases through more than one generation. In one remarkable series recorded by Barsickow, twenty-four cases were distributed through five generations, and the disease was also traced through five generations in a group described by Landouzy and Déjérine. It is very rare for the disease to be confined to one generation, far more rare than for pseudo-hypertrophic paralysis to be so confined. As in the case of most congenital hereditary diseases, cases that are apparently isolated are occasionally met with. Such isolated cases are rare; how rare we cannot at present say, because it has generally been the family tendency that has suggested the idiopathic nature of the disease, and it is probable that many isolated cases have been thought to be spinal, the more readily since the disease has little tendency to cause death, and its real nature, if not suspected during life, has seldom been discovered. That isolated cases occur is undoubted. In the families of the patients shown in Figs. 150-3 and 154 no example of analogous disease could be heard of.

Both sexes suffer, and it is doubtful whether either sex is the more prone to the disease. In a few families, females have suffered chiefly, in others males, but in most groups the individuals affected have been of both sexes. The age at which the disease first manifests itself is extremely variable. It may begin as early as three and as late as

sixty years. But the onset is rarely during childhood; in the majority of cases the disease shows itself between fifteen and thirty-five; that is, during the later period of growth and the early period of adult life. In the same family, the variations may be extreme. In that of which the history has been given by Barsickow the date of the onset of seventeen cases was known; in one the disease began at twelve, in four between fifteen and twenty, in seven between twenty and thirty, in three between thirty and forty, and in two after forty. Sex has no influence on the date of onset, nor, as a rule, can any relation be traced between the date and the place at which the wasting begins. When the wasting begins in the face, the disease more frequently commences in childhood than when the first wasting is in the limbs; but, in some instances, the atrophy has commenced in the face late in life, and this in the same family in which other sufferers have been young.* Thus, in the sexual proclivity, and in the date of onset, there is a marked difference between this disease and pseudo-hypertrophy; the latter showing a strong tendency to affect males, and to manifest itself in childhood.

As a rule no direct exciting cause can be traced. In a few instances the onset has succeeded some other morbid process,—such as chlorosis, acute disease, rheumatic affections due to exposure to cold,—and the depression of general health resulting from these may have determined the time of onset. But in other individuals of the same families the disease has developed without the aid of any exciting influence.

SYMPTOMS.—The onset is always gradual. Weakness and wasting come on together, and are noticed simultaneously, unless the commencing atrophy is concealed by subcutaneous fat. The atrophy generally begins in the upper arm muscles, the legs, or the face; the most frequent commencement is in the arms, but in some families there has been a tendency to commencement in the face, in others in the legs. The face was first affected in the lad figured on p. 406. In the part first affected the disease slowly increases, and thence it usually spreads. The onset may be symmetrical on the two sides, or one side may suffer some time before the other.

Of the arm-muscles the weakness and wasting are noticed first in the biceps and triceps, and with these the supinator longus often suffers. But examination generally shows that the lower part of the pectoralis and latissimus dorsi are greatly wasted, a loss of which the patient may be little aware, on account of the relative unimportance of these muscles. Sometimes the upper part of the pectoralis, and even the pectoralis minor, are also affected (Fig. 151). The tendency to atrophy of the lower part of the pectoralis and latissimus is a character common to this disease and pseudo-hypertrophic paralysis, but not invariable in either. The deltoids are rarely involved; in many cases they are

* It seems therefore undesirable to form a separate variety of "juvenile muscular atrophy," as Erb has proposed. Spinal atrophies may also be juvenile.

normal; sometimes they have been thought to be unduly large; in a few instances they have been wasted (Fig. 154). The serratus magnus is often affected (Fig. 153), but may escape, even in a severe case. The supraspinatus, infraspinatus, and subscapularis are rarely involved; the trapezius and rhomboids have been affected in many cases, sometimes entirely atrophied (Fig. 153).

The forearm-muscles generally escape, with the exception of the supinator longus, which usually wastes entirely when the other forearm-muscles are intact. Occasionally there has been some weakness of the long extensors or flexors of the fingers, with or without slight visible wasting. In the case mentioned on the next page, the extensors of the phalanges of the thumb were involved on the left, and the radial extensor of the wrist on the right side. Rarely the forearm-muscles have been much atrophied. In several instances there has been some atrophy of the small muscles of the hands, the thenar and interosseal muscles (as in a case presently to be described), or the interossei only, as in the case of Landouzy and Déjérine, but in the majority the integrity of these muscles was a marked feature of the disease, and a contrast to the spinal form.

The affection of the face is peculiar. There is commonly a failure of the zygomatic muscles, and in consequence a loss of the naso-labial furrow, and a curious alteration in the smile; instead of the angles of the mouth being drawn outwards and upwards, they are moved upwards by the elevators of the upper lip and angle of the mouth. The orbicularis oris is also affected, and in consequence the lips are habitually separated, the lower lip projects, the patient cannot "pout," and the articulation of labials is imperfect. The face has a dull expression and the aspect is very peculiar; it has been termed, by Landouzy and Déjérine, the "myopathic face." Rarely (as in Fig. 151) the frontales have been involved, and the orbicularis palpebrarum has been weak. In this case the affection of the face is unusually complete. Wasting of the muscles may be indistinct, because the contour of the face is only to a slight degree influenced by the substance of the muscles. The projecting lower lip may, indeed, appear to be thicker than normal. In many cases the buccinators have been affected, in some instances they have been normal, and then have drawn out the angles of the mouth in smiling. The tongue has been always unaffected, and so also have the pharynx, larynx, muscles of mastication, and the eyeball-muscles.*

* In a singular case under my care some years ago, an affection of the facial muscles, similar to that of idiopathic muscular atrophy, was associated with paralysis of ocular muscles. The patient was a girl twenty-seven years of age; there were no indications of syphilis, nor could any history of muscular atrophy in the family be ascertained. The ocular palsy commenced gradually at twenty-four, and increased until the movements of both eyes upwards, of the left eye inwards and the right outwards, were lost, and all other movements were weakened. The eyelids drooped slightly; the internal ocular muscles were normal. The affection of the face followed that of the eyes; the zygomatic muscles were powerless, so that the smile consisted

The muscles of the spine have sometimes been normal, sometimes they have been considerably atrophied. The intercostals are often affected in the later stages, but rarely in extreme degree. The diaphragm also sometimes suffers, as in the case shown in Fig. 150. The abdominal muscles have been involved in only a few instances. In the legs the muscles most commonly affected are the flexors of the hip, the extensors of the knee, and less frequently the glutei. The muscles below the knee have escaped in many cases; when they have suffered, the atrophy has generally involved the peroneal group, and the calf-muscles have been normal, a very marked contrast to pseudo-hypertrophic paralysis. The calf-muscles, however, shared the general wasting in the case shown in Fig. 154.

The electric irritability of the affected muscles is usually lessened in proportion to the wasting, and equally to both currents. Indeed, the diminution is often out of proportion to the wasting, and may be great when the atrophy is slight. There is no trace of degenerative reaction, and there is not even the longer persistence of voltaic than of faradaic irritability which occurs in the more chronic cases of spinal atrophy. Fibrillation is also absent, as an almost invariable rule; it has been observed only twice. Myotatic irritability is lessened or lost, it is never increased. Some shortening of muscles has been occasionally noted, frequently in the calf-muscles, rarely in the biceps.

All other functions of the nervous system are unaffected. Sensibility is normal. In a few cases transient rheumatoid pains have accompanied a rapid development of the disease in the arms, but in the vast majority of cases the disease is painless. The sphincters are unaffected, and there is no tendency to trophic or vaso-motor disturbance.

As the muscular atrophy progresses, the form of the affected parts becomes changed just as in spinal atrophy. Deformities may also occur, chiefly from the shortening of unaffected muscles, but these rarely reach the considerable degree common in other forms of muscular atrophy. Lordosis occurs in the upright posture, and is probably due to the same mechanism as in pseudo-hypertrophic paralysis, ceasing, as in that disease, when the patient sits (compare Figs. 150 and 153). When the muscles of the lower leg are involved, talipes may develop.

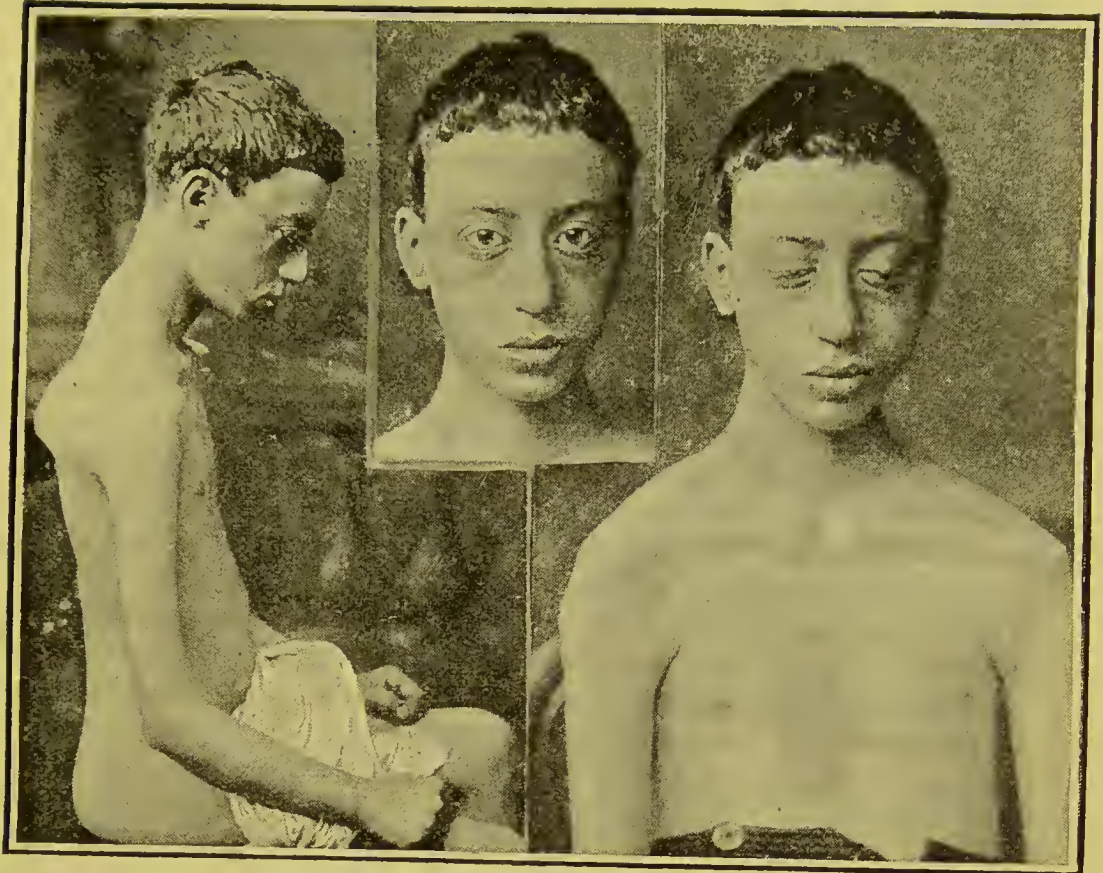
The course and duration of the disease are exceedingly variable. The atrophy may remain limited to the part in which it begins. The face alone has been affected in some members of a family, although in

only in elevation of the upper lip; the orbicularis was weak. The palate, pharynx, and larynx were normal. The arms became feeble, and the flexors of the hips almost powerless. There was no visible change in the nutrition or electrical irritability of the muscles; the knee-jerk was normal. If the case was central, as it appeared to be, the peculiar affection of the lips and zygomatics is not confined to idiopathic muscular atrophy. If the disease was muscular, the eyeball-muscles do not invariably escape.

FIG. 150.

FIG. 151.

FIG. 152.



FIGS. 150 TO 153.—Idiopathic muscular atrophy affecting the face. Fig. 150 shows the wasting of the deltoid, upper arm-muscles and supinator longus, and the rotation of the scapula from the loss of the trapezius and rhomboids. Fig. 151 shows the habitual appearance of the face, and Fig. 152 the greatest possible movement of the facial muscles in closing the eyes and smiling, and also the wasting of the deltoids and pectorals. The general distribution of the atrophy is seen in Fig. 153.*

FIG. 153.

* The patient is a lad, aged sixteen. No family history of any similar affection. He had primary and secondary syphilis at three. The wasting began gradually during childhood; movement of the face almost ceased at six; walking became difficult at twelve. Now, the face is almost motionless; lips full and can be brought together, but not shortened. Eyeballs prominent, their movements normal, and also those of masseters, tongue, pharynx, and larynx. Sterno-mastoids small, amohyoids large. Quite atrophied are the diaphragm, trapezii, rhomboids, deltoids, pectorales, latissimi, serrati, biceps, braehiales, triceps, supinators, the left extensors of the phalanges of the thumb, and right ext. carpi radialis. Unaffected are the other forearm and hand muscles, the lev. ang. seap., supra- and infra-spinatus, the last being large, probably from over-use. Erectors of spine feeble, intercostals strong. Extensors of hip and flexors of knees normal; flexors of hip and extensors of knee very feeble, the latter small; muscles of lower leg small and feeble, peronei powerless; some contraction of calf muscles. Knee-jerk absent. Electric irritability very low to each current in affected muscles; in most, no contraction to be obtained. Functions of nervous system normal.



other members the limbs subsequently suffered. In cases in which the wasting spreads, years may intervene before the extension takes place. In one case, for instance, the right arm became affected at nineteen, the left at twenty-five, the legs at thirty. In another the arms began to waste at thirty-five, power of standing was only lost at fifty-five, and the patient lived till seventy-five. Even when the malady begins in childhood its progress is sometimes very slow. Thus in one case the affection of the face was first noticed at five, at twelve the arms began to suffer, and a few years later the flexors of the hip became weak, but the patient was still able to walk at the age of forty. On the other hand, the progress of the disease may be more rapid and uniform, and the atrophy may reach its widest distribution in eight or ten years. In most cases, even of severe type, the wasting does not become universal, but remains limited to the muscles mentioned above. Occasionally hardly any muscles of the body may escape. An example of such rare universal atrophy is shown in Fig. 150, and the case is also noteworthy from the commencement in a somewhat rare seat, the lower legs, and in the degree of involvement of the hands. The patient was a man aged twenty-seven at the time of his death (in University College Hospital). No history of any analogous case in his family could be ascertained. The affection

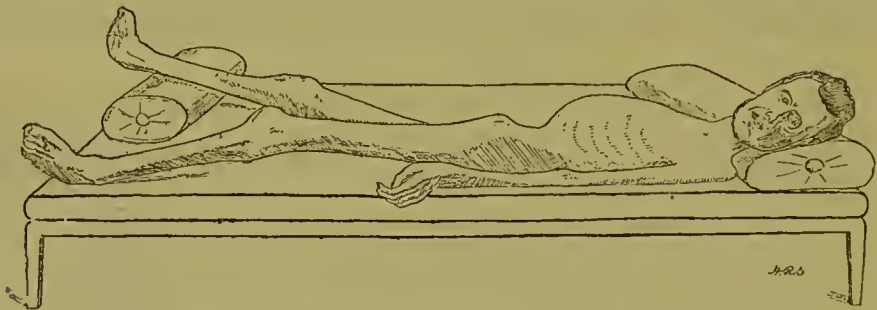


FIG. 154.—Idiopathic muscular atrophy. (Drawn by Dr. Spencer, from a photograph.)

began at the age of fourteen, when his feet began to turn in so that he walked on the outer side of the foot, and soon he noticed gradual wasting of the legs, which slowly progressed, and about the age of twenty-four the arms also began to suffer. When first seen, a few months before his death, the muscular atrophy was universal, and the subcutaneous fat had also disappeared. Even the hands were greatly wasted; there was a hollow in the position of the thenar eminence, just as in progressive muscular atrophy, and the interosseal muscles were also greatly wasted. There was slight fibrillation. The intercostals were paralysed. The wasting of the legs was extreme; the maximum circumference of the calf was only seven inches; the feet were inverted, the sole hollowed, and the toes flexed. The patient died from diarrhœa. The muscles were found to be small and pale. The chief microscopical change was intense granular and fatty degeneration of the fibres. A very few normal fibres were seen in some muscles.

There was no increase of interstitial tissue. Microscopical examination revealed no morbid changes in the spinal cord.

The duration of the disease varies from ten to fifty years. Death has never occurred as the direct result of the malady. In the cases of most severe degree and rapid course, the patient has usually died from phthisis, probably related to the deficient breathing power, just as in the sufferers from pseudo-hypertrophic paralysis. In most cases, however, death has been due to other maladies and has not been in any degree the consequence of the muscular disease.

PATHOLOGICAL ANATOMY.—The pathological facts are limited to two or three post-mortem examinations, and a few observations on excised fragments of muscles. The conditions found have varied much. In the case recorded by Landouzy and Déjérine, simple narrowing of the muscular fibres was the chief morbid appearance. In some cases excised fragments have shown considerable increase in the interstitial tissue; in the case shown in fig. 154, a granular and fatty degeneration of the muscular fibres was the only change. In some excised fragments, fibres have been seen larger than normal, but whether these were really hypertrophied, or merely thickened by contraction, is uncertain (see note on p. 411). Longitudinal striation and fissuring of the fibres has been described by Erb. The spinal cord and nerves, when examined, have been found normal. The condition is thus diverse, and it is difficult to conceive that they can all be results of the same cause, and manifestations of the same disease. But it is to be noted that in cases of the pseudo-hypertrophic type considerable diversity has been met with in the muscular changes. So far as our present knowledge goes, the muscular changes must be ascribed to a congenital tendency by which they perish prematurely. The tendency may lead to overgrowth of the interstitial tissue, or to simple wasting of the fibres, or to early decay, either in consequence of a simple difference in the characters of the tendency, or more probably by the co-operation with it of other constitutional influences, congenital or acquired.

DIAGNOSIS.—The two most important diagnostic indications are the affection of more than one member of the same family, and the onset of the disease before adult life is reached. The former is alone conclusive; but the latter should always suggest the probable idiopathic nature of the case. In isolated cases the diagnostic difficulty is much greater, and no indication is actually conclusive, except perhaps the affection of the face. Commencement during childhood is also of great weight. It is most unlikely that progressive muscular atrophy, beginning under ten, is of spinal origin, but I have known atrophy, almost certainly spinal, to commence at the age of fourteen. The peculiar affection of the zygomatic muscles is very characteristic, but we cannot yet say that it is pathognomonic (see note on p. 405), and the affection of the lips must be carefully distinguished from that due to the bulbar palsy so

commonly associated with spinal atrophy. Of the distribution of the wasting, that of the latissimus and lower half of the pectoralis, and the escape of the hand-muscles, are the most important characteristics, but they are not, by themselves, conclusive, since they are sometimes met with in spinal atrophy.

Other most important distinctions between spinal and idiopathic atrophy depend upon the symptoms of the former, which are absent in the latter. Among these are the affection of the tongue, palate, and larynx, the commencement of atrophy in the hands, and the excess of the knee-jerk which results from lateral sclerosis. The latter is a conclusive indication of spinal disease, and therefore renders the spinal origin of the atrophy all but certain. The distinction from the pseudo-hypertrophic form has been already considered in the account of this disease.

PROGNOSIS.—The extreme variations in the course of the disease render the prognosis in any individual case very uncertain. Unfortunately recovery from atrophy that has actually occurred cannot be anticipated, but on the other hand, the disease may remain limited to its first seat, or may not involve other parts until after many years. In a considerable number of cases, perhaps one half, the malady has not appeared to shorten life. Even in cases that begin during youth it is therefore possible that the patient may reach old age.

TREATMENT.—It is not yet clear that idiopathic muscular atrophy can be influenced by treatment in any considerable degree. Most published records of cases are almost silent on the subject, and the disease is so rare that few individuals have an opportunity of forming an opinion. It might be assumed that the essential cause of the disease, a congenital tendency, withdraws it from the range of therapeutics. But the course of the malady is very different from that of some other diseases which own a similar cause. The extreme variations in the date of onset, the fact that the disease may not be manifested until late in life, and the long period that may intervene between its onset and extension, all suggest that other influences co-operate with the congenital tendency in determining the development of the malady. It is quite possible, therefore, that the first inference from the history of the disease may not be altogether correct, although it must be admitted that we have not as yet any evidence that the disease is susceptible of influence from drugs. Electrical treatment and massage have been thought to do good and even to arrest the disease (Erb), but the variable tendency of the malady renders the evidence of arrest insufficient. If voluntary exercise is practicable, this is a far more efficient stimulus to muscular growth than any electrical application, and it is probable that such exercise, carefully persevered in, may do something to prevent the occurrence of the malady in those predisposed to it, and even to retard its progress in those who are

already attacked. In this connection it is noteworthy that very few of the sufferers have been engaged in occupations that involve active muscular exertion. Over-exertion should, however, be carefully avoided. The general health should be attended to, and any defect removed as speedily as possible, both in those who suffer and in those who are related to sufferers, since, as we have seen, depressing influences may apparently excite the development of the disease, and it is therefore reasonable to suppose that they may accelerate its progress, and lessen any tendency there may be for the morbid process to become stationary.

MUSCULAR HYPERTROPHY.

The occurrence of a true hypertrophy of muscles, as a condition of disease, is exceedingly rare. In most cases in which the muscles are enlarged, the increase in size is due to a growth of interstitial tissue, fatty or fibrous, such as has been described in the chapter on pseudo-hypertrophic paralysis. We have seen that muscular fibres larger than normal have been met with in that disease, and also in some muscles of normal or increased size in cases in which other muscles were simply atrophied. But this increase in size has been met with only in excised fragments, and it is possible that the condition may have been due to a vital contraction excited by the process of excision. The same ground of doubt exists in all cases in which a true hypertrophy has been described; the fact rests upon observations made on excised fragments, and has not yet been observed after death.*

The condition has been found in the singular malady described in the next section (Thomsen's disease). It has also been met with, in very rare instances, as a widespread or partial condition, usually associated, strange to say, with either diminished power or with a morbid readiness of fatigue.† The muscles most frequently affected have been those of the shoulder and upper arm, or of the thigh and calf, on one side or on both. It has also been observed in the glutei, spinal muscles, and trapezii. The causes are obscure, but it has been ascribed to over-exertion. The diameter of the fibres has been increased

* Since the above was written, the doubt there expressed has been confirmed by an observation made for me by Dr. H. R. Spencer. A fragment of the gastrocnemius of an amputated leg was excised immediately after the amputation, and the fibres were compared with those of the muscle twenty-one hours later. In order to ascertain more definitely whether a vital contraction persisted as an apparent increase in size, a part of the excised fragment was separated and faradised. The average size of the fibres in the muscle was $\frac{1}{40}$ inch, in the fragment excised $\frac{1}{25}$ inch, in the fragment faradised $\frac{1}{17}$ inch. Since the division of the nerve during the amputation may have caused some contraction, it is possible that the difference in size produced by excision may be even greater than these figures represent.

† Auerbach, 'Virchow's Archiv,' Bd. 53, pp. 234 and 397; Berger, 'Deut. Archiv f. klin. Med.,' Bd. 9, 1872, p. 363; Friedreich, 'Ueber Prog. Muskelatrophie, &c.,' 1873; Eulenberg, 'Real-Encyclopädie,' Bd. ix, p. 354.

to double the normal size, a maximum of $\frac{1}{200}$ th inch having been met with (Eulenberg), whereas the normal maximum may be taken as $\frac{1}{400}$ th inch. An increase of the nuclei has been observed, without any overgrowth of the interstitial tissue.

The condition is manifested by an increase in the size of the muscles, which are also firm. The circumference of the limb is greater than normal, and when the change is unilateral, the difference between the limbs of the two sides may be very striking. The maximum circumference of the calf has been as much as seventeen inches. The muscles are soon exhausted, and have been weak in some cases, while in others there has been abnormal strength for brief exertion. The electrical, mechanical, and myotatic irritability of the muscles has usually been found normal.

The disease may be suspected if a marked increase in size in an adult is accompanied by impaired power of sustained exertion; if the muscles are firm, and the patient does not manifest other indications of pseudo-hypertrophic paralysis. The diagnosis can, however, only be made with certainty by the microscopical examination of an excised fragment. The meagre facts regarding the course of the disease suggest that it usually persists without getting either better or worse. Treatment appears to have but little influence upon it.

A singular case has been reported by Eulenberg,* which differs in many particulars from the form of disease above described and illustrates the complex relations of enlargement of the muscular fibres and its connection with degenerative processes. A man aged thirty-six presented an enormous enlargement of the muscles of the left leg, which were soft and flabby and weak, with lowered irritability. The condition had slowly developed after a fall on the back at ten, which caused imperfect paraplegia, motor and sensory. A year later, during pleurisy, he had thrombosis of the left femoral vein. In an excised fragment of the gastrocnemius the muscular fibres were large, the maximum being $\frac{1}{75}$ th of an inch, and presented fatty and vitreous degeneration, and fat was seen between the fasciculi. The muscles of the other leg were somewhat wasted, but the fibres were also enlarged and degenerated. It would appear as though the condition had resulted from a traumatic lesion of the spinal cord, and had been intensified in the left leg by the influence of the thrombosis and resulting vascular disturbance.

* 'Deutsch. Med. Wochenschrift,' 1885, No. 12.

THOMSEN'S DISEASE.

The malady thus designated may be considered here, notwithstanding the obscurity of its nature, because it has at least these points in common with the diseases last described, that the symptoms are muscular and that the disease is commonly congenital and occurs in families. It has been named after the physician whose description gained for it general notice, and who is himself its subject, but it had been previously described by Leyden, and hinted at, long ago, by Sir Charles Bell.*

The disease is characterised by a peculiar rigidity of the muscles, which comes on when they are first put in action after a period of rest. The rigidity is transient, and when it has passed off it does not return as long as exertion is continued. The malady is usually congenital and often hereditary, affecting many members of the same family. In that of Thomsen, cases can be traced through five generations. It appears to be more common in Scandinavia and Germany than in France or England, but it is a rare disease; only about thirty instances have been recorded, and it is probable that these are all the cases in which the affection has been recognised. At the same time there is little to attract attention in the slighter forms of the disease, and it may ultimately be found to be less rare than at present it appears to be.

Both sexes suffer. In most cases the symptoms have been first noted in early childhood, at four or five years of age, sometimes even in the cradle. They increase during the period of muscular development, and then remain stationary. In a few instances the patient has seemed free until about the period of puberty, but even in such instances it is probable that the disease is congenital in origin, although its manifestation is deferred, since the cases have occurred in families, other members of which have suffered earlier. But in a few cases symptoms apparently identical with those of the congenital disease have come on in early manhood after some exciting cause, in one case a lightning stroke, in another a sudden alarm.† In such cases the disease has been apparently acquired.

The characteristic symptom is tonic spasm of the muscles when they are put in action after a period of rest. As soon as the patient attempts to move, the muscles become rigid. The rigidity may make

* The most important papers on the disease are those of Leyden, 'Klinik der Rückenmarkskr.,' 1874, Bd. i, p. 128; Thomsen, 'Arch. f. Psychiatrie,' Bd. 6, 1876, p. 702, and 'Centralbl. f. Nervenkr.,' 1885, p. 193; Bernhardt, 'Virchow's Archiv,' Bd. 75, 1879, p. 516, and 'Centralblatt f. Nervenkr.,' 1885, p. 122; Ballet and Marie, 'Archives de Neurologie,' 1883, No. 13; Möbius, 'Schmidt's Jahrb.,' Bd. 198, 1883; Ringer and Sainsbury, 'Lancet,' 1884, pp. 767, 816, and 860. The mention of the symptoms by Sir Charles Bell is at p. 436 of his 'Nervous System,' Case 184, "Affection of the Voluntary Nerves."

† Engel, 'Phil. Med. Times,' 1883, p. 412; Schönfeld, 'Berlin med. Wochenschr.,' 1883, No. 27.

movement impossible, while it lasts, or may merely lessen the possible range of movement. After a few minutes or less the spasm passes away, to be renewed, but in slighter degree, by a fresh attempt, and if the movements are persevered in, the spasm, in a few minutes, becomes trifling, and does not return until after rest; the individual can walk for hours without fatigue when the spasm has passed off. The rigidity is always increased by attention and by nervous apprehension of it, and the more the sufferer tries to overcome the stiffness, the less is he able to do so. In a severe case, a slight impulse will make the person fall, and it may then be impossible for him, for some minutes, to rise from the ground. The arms are usually less affected than the legs, but in some cases the rigidity fixes the fingers for a short time on an attempt to use the hand. The muscles of the face are usually free; mastication may, however, be interfered with by the spasm in the muscles of the jaw. Rarely the tongue has been affected; still more rarely the muscles of the eyeball. Sometimes the spasm is greater on one side of the body than on the other.

The muscles are always well nourished; they are often, indeed, above the normal size, and possess more than normal strength. Thomsen believes that the more the muscles are habitually used, the less severe is the spasm, and that a life of active exertion produces some permanent amelioration in the disease. It is, however, a source of some disability, and of considerable annoyance; in the words of Thomsen, "it casts a shadow over the lives of the sufferers."

Careful investigations of the muscular phenomena have been made by several observers, and that by Erb is especially instructive. In his case a single brief effort caused tonic contraction which lasted for twenty-five seconds. Momentary electrical stimulation of the nerves caused only a momentary contraction of the muscles, but continued stimulation caused a prolonged contraction similar to that excited by the will. In certain muscles moreover an unbroken current caused peculiar wave-like contractions, about one per second, passing from the negative to the positive pole. Any strong stimulation of the muscle itself caused a similar after-contraction. The irritability of the nerves was normal, but the voltaic excitability of the muscles was unduly great, and they were remarkably sensitive to mechanical stimulation. Firm pressure caused a tonic contraction lasting from twelve to twenty seconds.

PATHOLOGY.—Of the morbid anatomy of the disease we know only that the muscular fibres, in excised fragments, have been normal in aspect in some cases, but in others, especially that observed by Erb, many fibres have been seen exceeding the normal maximum in size. The minimum fibres were the same size as in health, but the largest were twice as large as any met with in normal muscle.

The manifestation of the disease is in a disorder of the functions of the muscles, and most writers on the subject have followed Leyden in

regarding the malady as essentially muscular in nature, as consisting in an altered functional condition of the muscular tissue. This opinion is strongly supported by the frequent alteration in electrical excitability, which can hardly be otherwise interpreted than as an indication of a change in the mode of action of the contractile muscular protoplasm. This opinion is supported also by the interesting experiments of Ringer and Sainsbury, who found that certain salts, such as sodium phosphate, are capable of causing in the frog tonic spasm bearing considerable resemblance to that of Thomsen's disease, and that such spasm persists, not only after the nerve has been divided, but after the intramuscular nerve-endings have been paralysed by curara. Although this fact does not, of course, prove that Thomsen's disease is muscular in nature, it shows that a similar condition may have its origin solely in the muscles, and it harmonises with the indications afforded by the muscular irritability in this disease. But a proof that there is a derangement of the function of the muscles is no proof that this is the sole element in the disease, and that it is not is at least suggested by two other facts. First the disease has apparently resulted, in rare cases, from morbid influences acting on the nervous system in adult life, and secondly, in the congenital cases, the spasm is increased by emotion. Wide as is the difference between the muscular and nervous tissues, we must remember that they have some conditions in common. The dependence of the nutrition of the muscles on that of the motor nerve-fibres and cells is a very remarkable fact, and so also is the influence of the functional activity of the cells and fibres in causing a similar condition in the muscular tissue. To say that the two structures are connected is hardly an explanation of the fact. Whatever is the nature of the relation between them it is at least conceivable that an abnormal functional state, congenital in nature, may be common to the two, and that the peculiar over-action in the muscles may be accompanied by a similar over-action in the ganglion cells of the cord, and even in the pyramidal cells of the cortex. It is even conceivable that the condition of the nerve-cells may be the primary change, and that of the muscles may be secondary, although, when produced, it is in some degree, independent, and may be excited independently by local stimulation. Such a theory enables us to understand the two facts above mentioned, the influence of emotion and the acquisition of the disease, each of which seems to be inconsistent with a purely muscular pathology.

TREATMENT.—No treatment, properly so called, appears to exert any influence on the disease. The congenital malady persists through life; in the cases in which a similar condition has apparently been acquired, it has also been persistent. The only influence that has appeared to Thomsen to ameliorate the condition is a life of active muscular exertion.

Under the name of *Congenital Paramyotone* Eulenberg has described* a strange family affection allied to Thomsen's disease in its general character, although differing very much in its special features, and equally obscure in nature. The malady was widely spread in the affected family, and could be traced through six generations, but appears now to be dying out. Its congenital character was shown not only by its multiplicity, but also by the fact that in some individuals it was manifested immediately after birth. The symptom was tonic spasm, lasting from a quarter of an hour to several hours, excited chiefly by cold although often by merely slight cold. The rigidity was followed for a time by weakness. The facial muscles were very prone to become thus rigid, especially the orbiculares palpebrarum and oris, and while the contraction lasted the patient was often unable to speak or to open the eyes. The rigidity was slighter in the legs than in the arms, but the subsequent weakness was equally marked. Warmth removed the spasm. There was no persistent loss of power, and no increase of mechanical irritability. The electrical excitability of the nerves was normal; that of the muscles was lowered to each current, and there was an abnormal tendency to tetanic contraction during the passage of the current. Eulenberg speculates that the symptoms may be due to reflex vaso-motor spasm in the muscles, because the diminution of the blood-supply to muscles renders them weak, but it is clearly equally possible that the two certain phenomena, the sensory impression, and the muscular contraction, may be directly connected.

TUMOURS OF THE SPINAL CORD.

Morbid growths within the spinal canal may spring from the membranes or may grow in the substance of the cord itself. The difference in seat entails some difference in symptoms, but it is nevertheless convenient to consider the two classes together, because they have many symptoms in common, and it is often impossible to carry the diagnosis farther than the existence of an intraspinal tumour.

ETIOLOGY.—Of the diathetic conditions which give rise to tumours elsewhere, only two, syphilis and tubercle, are effective in causing growths which commence with the spinal canal. Parasitic tumours occur, due to the same influences which produce them in other situations. A few rare growths appear to be congenital in origin, due to the development of germinal tissue in an abnormal position. Of the causes of other forms of tumour we know practically nothing. Injuries, such as a blow on the spine, have been supposed to be occasional

* 'Neurologisches Centralblatt,' 1886, p. 265.

causes, and their influence in rare instances has seemed possible, but the evidence is not so strong as it is in the case of tumours in some other situations. In many cases the first symptoms have immediately followed some exposure to cold and wet, and it seems probable that this influence may have excited secondary processes in the nerve-elements which were already deranged by the growth, but it can have had no share in the production of the growth itself.

PATHOLOGICAL ANATOMY.—The growths within the spinal canal may develop outside the dura mater, inside the dura mater, or within the substance of the cord. The extra-dural tumours may spring from the membrane, or from the tissue between the membrane and the bone, or may grow into the canal from the outside, through the intervertebral foramina. Subdural tumours may proceed from the inner surface of this membrane, from the arachnoid, or from the pia mater (Fig. 157). The growths in the cord may spring from the pia mater, or may develop in the substance of the cord. They sometimes proceed from the peculiar tissue which surrounds the central canal.

The forms of extra-dural tumours are lipoma, from an overgrowth of the fat which normally exists between the membranes and the bone, and parasitic tumours, chiefly echinococci. Both are rare. Growths may also spring from the bones or intervertebral tissue, enchondroma, sarcoma, and cancerous tumours, but these have been already considered. Far more frequent are collections of inflammatory products from bone-disease, but these do not come into the category of morbid growths.

The tumours which begin within the dura-matral sheath are chiefly syphilomata, sarcomata, and myxomata, sometimes containing cysts or "brain-sand." Tubercular and parasitic tumours are rare, but both echinococci and cysticerci have been met with, developing in the meshes of the arachnoid. Fatty tumours have been found in a few instances. Fig. 155 represents a probably unique tumour, a myo-lipoma, consisting of fatty tissue and striated muscular fibres, which had grown from the pia mater or meshes of the arachnoid, and was no doubt congenital. Neuromata may occur on the nerve-roots, and may compress the cord.

The growths within the substance of the cord itself are more diverse in character. Syphiloma and glioma are the most common; sarcomata, myxomata, and tubercular tumours also occur. A cysticercus has once been met with. Some growths have a compound character, and have been termed myxo-sarcoma, glio-sarcoma, and fibro-sarcoma. Sarcomata and gliomata are sometimes very vascular and such growths have been termed "angio-sarcoma" and "angio-glioma." It is far more common for the tumour to spring from the pia mater or from the peri-ependymal tissue around the canal, than for it to begin among the nerve-structures.

Extra-dural growths are always single; those within the dura mater are often single, but sometimes two or three coexist, and neuromata

or sarcomata on the nerve-roots are often multiple. Tumours within the spinal cord are also usually single, but occasionally more than one growth is found, especially when the tumours are tubercular.

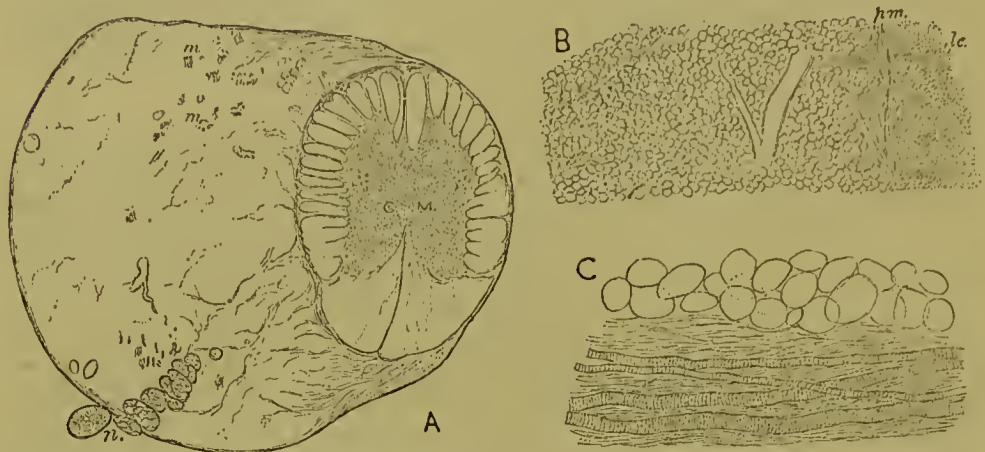


FIG. 155.—Myo-lipoma of the spinal cord. A, transverse section of the tumour and of the conus medullaris of the spinal cord, to which the growth was attached, and of which the grey matter is represented by the dotted shading C M. *n.*, sections of nerve-roots, partly enclosed in the tumour; *m.*, bundles of muscular fibres. B, part of the tumour more highly magnified, showing the fat cells of which it was chiefly composed; *p. m.*, pia mater of the cord, of which *l. c.* is part of the lateral column. C, part of the tumour, still more magnified, showing striated muscular fibres, fibrous tissue, and fat cells (see also Fig. 149, p. 398). The tumour had caused no symptoms.

The size attained by growths outside the cord is necessarily moderate in consequence of the limitation of the space in which they grow. They vary from the size of a pea to a width of one and a length of two inches, which is rarely exceeded, and chiefly by extradural tumours. Those that develop from the pia mater are usually smaller than those that spring from the dura mater. Multiple tumours on the nerve-roots are generally small. Adhesions often form between the growth and the membrane from which it does not arise. The tumour compresses the nerve-roots and the spinal cord (Figs. 156, 159). The compression produced by tumours within the dura mater is always greater than that by tumours outside this membrane. The amount of pressure is proportioned to the size of the tumour and its consistence. A soft growth outside the sheath may attain a considerable size, and even extend through the intervertebral foramina, without exerting much pressure on the cord itself. At the compressed part the cord is narrowed and softened, usually indented or flattened, because the pressure is either from one side, from the front, or from the back. Rarely the degree of pressure is so great that the cord is narrowed to the size of a crowquill, and it has even been apparently interrupted at the spot, the upper and lower portions being conical and their pointed extremities connected only by membranous tissue. The softening of the cord depends on inflammation, the "pressure-myelitis" described in a preceding chapter (p. 246), and it presents

FIG. 156.



FIG. 157.



FIG. 158.



FIG. 156.—Tumour growing from the inner surface of the dura mater, and compressing the spinal cord in the mid-dorsal region. The tumour, 3 cm. long, was a sarcoma in which the cells were arranged in concentric groups, the centres of which had undergone calcification. The spinal cord was softened and dark in tint at the compressed part.* (After Lancereaux.)

FIG. 157.—Sarcoma growing between the arachnoid and pia mater in the mid-dorsal region of the cord. The tumour had sprung from the meshes of the arachnoid, and was only slightly adherent to the pia mater. The spinal cord was compressed and softened. In the figure the dura-matral sheath has been laid open, except at the top, and the arachnoid has been opened over the lower half of the tumour which, 5 cm. long, lies on the posterior and (right) lateral aspect of the cord.† (After Lancereaux.)

FIG. 158.—A tumour (T) growing from the dura mater, and compressing the right side of the spinal cord at the origin of the 4th, 5th, and 6th cervical nerves. The tumour was a spindle-celled sarcoma springing from the arachnoid.‡ (After Leyden.)

* The patient was a woman seventy-one years of age, paraplegic, with strong flexor contracture of the legs, loss of power over the sphincters, and considerable impairment, but not absolute loss, of sensibility. The symptoms came on gradually six years before death. (Lancereaux, 'Atlas d'Anat. Path.,' p. 444.)

† The patient was a woman twenty-eight years of age. The first symptom was pain radiating over the upper part of the abdomen on the right side, and afterwards extending to the leg and to the left side. After a few months, weakness of the legs gradually came on, and became absolute, with loss of sensation and of power over the sphincters. Extensive bedsores were the immediate cause of death, which occurred eighteen months after the onset. (Lancereaux, 'Atlas,' p. 447.)

‡ The patient was a man aged thirty-five. At twenty-eight pain commenced in

FIG. 159.



FIG. 160.



FIG. 161.



FIG. 159.—Tumour of dura mater opposite the upper part of the lumbar enlargement, compressing nerve-roots and spinal cord. (From a preparation in University College Museum. Drawn by Dr. H. R. Spencer.)

FIG. 160.—Tumour of the cauda equina surrounding and enclosing many of the nerve roots. The cord itself was unaffected. The structure of the tumour was that of a fibro-sarcoma.*

FIG. 161.—Neuromata of the cauda equina. (After Lancereaux.) The growths had apparently given rise to no symptoms.

the right forearm, and continued, intermitting, for some years, until, at thirty-four, it extended through the whole arm to the neck beside the spine, where it was increased by movement. Similar pain then came on in the left arm. Then followed weakness in the right arm, slight spasm in the right leg, and tingling in the left. The arm became almost powerless and wasted, the right leg weak, and flexion of the neck caused severe local pain. These symptoms continued and increased, and pain in the left leg came on. The cervical spine became tender, and movement of the head to the right was limited. Then both legs became weak, and sensibility was lost as high as the nipples. The paraplegia became absolute, bed-sores formed, and the patient died seven years after the onset of the symptoms. Softening of the spinal cord extended down into the dorsal region. (Leyden, 'Klin. der Rückenmarkskr.,' Bd. i, p. 450.)

* The patient was a man aged twenty-eight, under the care of my colleague, Dr. Radcliffe, in the Queen Square Hospital. The symptoms commenced only nine months before death, and consisted in severe pains in the legs and progressive weakness, the power of standing being lost about four months after the onset. A little power in the flexors of the hips and the extensors of the knees persisted almost to the last, but the knee-jerk was lost. All the muscles of the legs wasted, those below the knee extremely, and even when the patient was first seen electrical irritability was greatly lowered to both currents, without any reaction of degeneration. Tactile sensibility was impaired in each foot and lower leg, more in the right than in the left, and chiefly in the region supplied from the sacral plexus. Sensibility to pain was not affected. The bladder was affected early, and, from imperfect evacuation, cystitis and symptoms of pyelo-nephritis already existed, a catheter having never been passed. The kidney disease continued, in spite of local treatment, and was the immediate cause of death, which was preceded by several convulsions, apparently

the tissue changes that have been there enumerated. As a rule the spinal cord is not invaded by a growth that develops outside it, even if this begins in the pia mater.

The growths that involve the cauda equina often attain a larger size than those that occur higher up, because this part of the vertebral canal is large, and the nerve-roots occupy but a small part of it. Most tumours in this situation are sarcomata or fibro-sarcomata. They usually spring from the tissue of the arachnoid and often surround and include the nerves (Fig. 160), the amount of damage to which is very variable.

Multiple tumours outside the cord are sometimes very numerous. They are usually sarcomata and spring from the membranes and sheaths of the nerves. They are various in size, ranging from that of a hazel nut to that of a pin's head, and many very small growths are often scattered among the nerves of the cauda equina. In some cases of this character similar growths have existed in the cerebral membranes.

The tumours within the spinal cord are usually small, seldom exceeding half an inch in diameter, and usually less, even when they give rise to considerable impairment of function. But their vertical extent often exceeds their transverse diameter, and sometimes they grow through a considerable extent of the spinal cord. Gliomata and growths that spring from the central tissue are those which have most frequently a considerable vertical extent. A glioma has been known to reach from the medulla oblongata to the lumbar region. The cord is enlarged at the seat of the tumour, and the precise character of the enlargement depends on the position of the growth. In an infiltrating glioma the increase in size has been so great that the wall of the foramen magnum caused a constriction around the swollen cord. Often the abnormal colour of the tumour is perceptible on the surface. The consistence of the cord may be lessened in soft growths, but the tumour is commonly firmer than the normal cord, and the difference is frequently increased by softening in the vicinity of the growth. If the tumour reaches the surface, the pia mater and arachnoid may be thickened at the spot, and they may even be adherent to the dura mater, but there is rarely any extension of the meningitis to other parts. On section, the growth is usually very distinct, since its aspect contrasts with that of the nerve-substance. Only glioma and myxoma may resemble the normal grey substance in appearance, but the new tissue is distinct in position. These tumours in some cases blend with the substance of the cord, but in other cases they are bounded by an area of softening which often isolates even invading growths. Other tumours may be sharply limited. It may be difficult

uræmic. A microscopical examination showed considerable damage to many nerve-roots involved in the tumour, while others had escaped. The muscles presented intense granular degeneration with some longitudinal striation, and increase of the interstitial nuclei.

to say in what structure the tumour began, but this may sometimes be ascertained by an examination of the upper and lower parts, when the

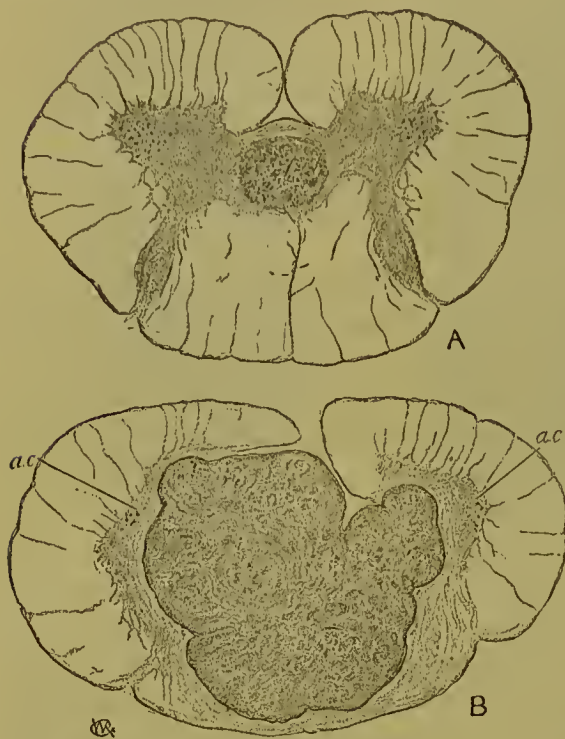


FIG. 162.—Tumour of spinal cord, springing from the tissue around the central canal. A, upper part of the tumour in the middle of the cervical region; the growth is here within the posterior commissure. B, section through the largest part of the tumour, which occupies the whole central region of the cord. The vertical extent was 2 cm. It was a sarcoma composed chiefly of spindle cells arranged in fasciculi and in concentric layers. The patient was suffering also from a cerebral tumour causing hemiplegia, and the spinal growth, which apparently developed rapidly during the final period of palsy and prostration, was not suspected during life.

growth is commonly limited to that part of the cord in which it first originated. Thus in Fig. 162, B, the tumour occupies a very large area, although the nearly equal extent on each side suggests its central origin, and this is clearly seen in a section through the upper part of the growth, in which the posterior commissure can be traced in front and behind the small area of growth, pointing to the peri-ependymal tissue as its source. Syphilitic tumours commonly originate from the pia mater, as they do also in the brain, and the membrane in their vicinity is often thickened.

They both invade and compress the nerve-tissue. An instance of such a gumma of the cord is shown in Fig. 163. It probably commenced at the furrow opposite the head of the posterior cornu, which it has destroyed, and the pressure which it exerted is evident from the displacement of the posterior median septum. In all forms of tumour

the central canal is often obliterated by the pressure and may be slightly dilated higher up, and this dilatation is especially common from growths that begin in the peri-ependymal tissue. The considerable dilatation of the canal which is termed "syringo-myelia" is associated with a peculiar growth in this position, probably congenital in origin; it is described in another chapter. In other forms of tumour cysts occasionally develop, and hæmorrhage may occur into such cysts, or into the softened tissue in the vicinity of the tumour, or even in the substance of a soft growth itself, especially when this is a glioma. From the region of the growth, secondary degenerations can frequently be traced upwards and downwards, but these are often slighter than the apparent damage would suggest, probably by reason of the

remarkable tolerance of the nerve-fibres to pressure if this is slowly developed.



FIG. 163.—Syphiloma in the spinal cord, occupying the position of the posterior cornu and adjacent parts of the posterior and lateral columns, in the lower cervical region. The tumour presented the usual combination of grey translucent, and yellow cheesy areas. A second similar, but much smaller, tumour existed higher up. The symptoms were complicated by hemiplegia, &c., of cerebral origin, due to a gumma in the brain. Paralysis with rigid flexor contracture of the left arm and leg were apparently due to the growth. The disease was certainly syphilitic, but it is noteworthy that it had developed during continuous treatment by full doses of iodide, which had entirely removed earlier symptoms, but to which the system had apparently become accustomed.

SYMPTOMS.—The symptoms produced by tumours within the spinal canal, whether these are within the cord or outside it, resemble in their general characters those which we have already considered as resulting from growth and caries in the bones of the vertebral column. The resemblance is most close in the case of extra-dural growths, and least so in that of tumours within the substance of the cord. The chief differences are the relative preponderance of symptoms of irritation of the cord itself, and the indications of a transverse extension of the damage from one side of the cord to the other.

In the majority of cases pain is a prominent symptom through the whole course of the disease. It is usually the earliest symptom, and is severe both before and after the development of other symptoms. The pain may be felt along the course of the nerves which arise at the level of the tumour and also in the parts supplied by nerves below that level, *e. g.* in the legs. Both kinds of pain are frequently very severe, darting, stabbing, rending in character. Dull aching pain may be felt between the attacks of severe pain. The intensity of the suffering is usually so great that it has more than once led the unhappy sufferer to

attempt suicide. The pains are commonly felt on one side first, in one arm, in one side of the trunk at a given level, or in one leg, and a considerable time may elapse before they extend to the other side. Occasionally the pain is bilateral from the first. The pains are sometimes increased by movement, but rarely in the intense degree common in tumours of the bone. Pain is sometimes felt in the spine; frequently when the growth begins in the dura mater, rarely when it is within the substance of the cord. Tenderness of the spine often exists in the former cases, but is on the whole a rare symptom. Other subjective sensations may occur with the pain or in the intervals; "numbness," tingling, formication, &c., and they give significance to the pains as an indication of organic damage to the nerve-structures. The root-pains in the trunk are often accompanied by a sense of constriction, which may be very distressing. Hyperæsthesia of the skin often accompanies the pains felt at the level of the tumour, less commonly those in the parts below.

Muscular spasm is another common symptom, most pronounced when the tumour springs from the membranes. There may be some rigidity of the back opposite the seat of the growth, usually associated with local pain. It is most marked when the disease is at the more mobile parts of the spine, especially when it is in the cervical region. In such cases the rigidity may be painful, and the pain may be increased by movement. Severe spasm in the abdominal muscles is often associated with severe girdle pain. Contractures often develop in the limbs, both in those supplied by the nerves which arise at the level of the tumour, and are directly irritated by it, and also, although less commonly, in the parts supplied from the spinal cord below the level of the growth. Thus the tumour in one half of the cord in the cervical region (shown in Fig. 163) caused persistent flexor contracture in the arm and leg on the side of the growth.

Paralysis is almost invariably one of the symptoms produced by an intraspinal tumour. Its onset is usually gradual. Paraplegia is the most common form, but all four limbs may be paralysed by a growth in the cervical region. Very commonly one leg becomes weakened before the other, and occasionally arm and leg suffer before the other side. Tumours which are situated in the middle line or in the centre of the cord, however, usually affect both sides at the same time. The palsy, gradual in onset, is usually also slow in its extension, the rate of its increase depending on the rate at which the tumour grows and compresses the spinal cord. This is the characteristic course of the paralysis produced by a tumour. But in a large number of cases the loss of power comes on in a subacute, or even an acute manner, in consequence of the myelitis excited by the compression, which, in such cases, may run a course of independent acuteness. When the inflammation is thus in excess of its cause, the palsy may lessen as damage from the inflammation subsides to the degree of the damage from pressure. The symptoms that depend on compression slowly

but surely increase as the tumour grows, and usually attain a high degree of intensity, the loss of power in the legs, for instance, becoming absolute and remaining so.

Loss of sensation comes on sometimes with the loss of motor power, more often after this has become considerable. It may be partial at first, but tends to become complete as the damage to the cord increases. The loss of sensation corresponds in distribution to the motor palsy when the disease is below the middle of the dorsal region, but if it is situated above this, and on one side, the sensory loss is often on the side opposite to the motor paralysis. Tumours of the spinal cord furnish a considerable proportion of the cases in which the crossed symptoms of a unilateral lesion are well marked (see p. 157). In addition to the loss of sensation which results from the damage to the cord itself, areas of anæsthesia may exist in the region supplied by the nerves which are damaged by the growth, and in which the severe radiating pains are felt. If there is crossed sensory and motor palsy, these root symptoms are intense chiefly on the side opposite to the anæsthesia in the limbs. Inco-ordination of movement is not a common symptom of spinal tumours, but has been occasionally met with in cases of growth in the posterior column, of central tumour, and also as a result of multiple tumours of the nerve-roots (see p. 428). Loss of power usually coexists and quickly becomes predominant.

The condition of reflex action depends on the position of the tumour. In the lumbar enlargement or the cauda equina, a growth abolishes reflex action in the legs, and in any position the trunk-reflexes are lost at the level of the lesion. But when the disease is situated higher up, reflex action is increased and the excess of cutaneous reflex action is a very marked and early symptom of the disease. The myotatic irritability presents also the increase that invariably results from damage to the pyramidal tracts, and the usual tonic spasm also gradually develops and often reaches a high degree. Muscular contracture is added to it more frequently than in many other diseases, and rigid spasm in the flexors of the knee and hip may keep the heels in contact with the nates.

Wasting of the muscles is for the most part confined to those parts to which the damaged nerve-roots go. In tumours of the lumbar enlargement and cauda equina, the atrophy of the muscles of the legs is a very conspicuous symptom. Vaso-motor disturbance sometimes occurs in the early stage of the disease, chiefly in the distribution of irritated roots, where flushing of the skin has been noted, and the so-called "meningitic streak" ("*tache cérébrale*") may often be produced. Occasionally vaso-motor œdema is a conspicuous early symptom. In the later stages, bedsores are very common and often severe. Dilatation or contraction of the pupil is common when a tumour is situated in the lower cervical region.

The course of the symptoms is variable, and depends on the rapidity

of growth of the tumour, and on the share which secondary myelitis takes in the production of the symptoms. As a rule the root symptoms occur first, and in growths outside the cord they may exist alone for months, and even, in the case of a slowly growing tumour, for years before the symptoms of damage to the cord itself are added. On the other hand, they may be entirely absent in growths which commence within the cord itself. In some cases the symptoms are steadily progressive, from first to last. In others the progress is intermittent; periods of increase in the symptoms alternate with stationary periods. Occasionally a rapid increase of the cord symptoms may be followed by positive improvement for a time, but scarcely ever by an actual disappearance of the manifestations of the disease. The most important element in the course of the disease is the tendency to a lateral extension of the symptoms, the expression of the transverse extension of the damage, as the several structures of the cord, at the same level, successively suffer. There is sometimes an extension downwards of the interference with the central functions of the cord, when secondary myelitis descends to the lumbar enlargement, but there is scarcely ever an extension upwards above the level of the initial interference with function. A small, slowly-growing tumour, even within the cord, sometimes causes no symptoms.

The chief differences between the symptoms of tumours of the cord and of the membranes are that, in the latter, the root symptoms are more severe in a larger proportion of the cases, and that the cord symptoms occur later, and are at first more limited in extent. Spinal pain, tenderness, and rigidity are more common. In cord tumours the radiating pains, due to irritation of the nerve-roots, are more often trifling or absent, but if the growth begins in the neighbourhood of the posterior horn they may be very pronounced. The cord symptoms are often bilateral from the first, and in central tumours they may develop equally on the two sides. If they are unilateral at the onset they extend to the other side sooner than in the case of meningeal growths. Extensive muscular atrophy is more common in tumours of the cord than in those of the membranes (except in the case of tumours of the cauda equina), because central tumours may damage the grey matter through a considerable vertical extent, and are most common in the enlargements, and also because myelitis spreads in the grey matter far more frequently when the exciting growth is in the cord itself than when it is in the membranes.

In tumours of the cervical region the interference with movement of the neck, from the rigidity of the muscles, is often well marked, especially when the tumour is in the membranes. The pains due to irritation of the nerves are felt in the arms, and are often associated with muscular atrophy. The excess of reflex action in the legs is very great, and if the influence of the tumour is chiefly on one side, hemiplegic weakness, with crossed anæsthesia, is present.

Growths in the dorsal region of the cord usually cause severe

radiating pains in the trunk, often accompanied by an intensely painful sense of constriction, by hyperæsthesia, and sometimes by anæsthesia. The simple, nearly level course of the dorsal nerves, reveals the precise position of the disease more clearly than do the symptoms in the arms when the tumour is in the cervical region. The interference with the trunk-reflexes often corroborates the indications afforded by the subjective symptoms. Spinal tenderness may be also present, but rigidity of the spinal muscles is not common. Reflex action in the legs presents the same excess that results from tumours in the cervical region, but occasionally, when the growth is on the confines of the lumbar enlargement, a descending myelitis may abolish the reflex action, and remove spasm, if this previously existed, rendering the muscles flaccid.

The symptoms produced by tumours which involve the lumbar enlargement vary considerably according to the precise position of the growth. The characteristic pains are felt in the legs, and the muscles supplied from the part invaded or compressed undergo atrophy and present loss of reflex action and of myotatic irritability, but the anæsthesia is often limited in area. A growth in the upper part of the enlargement may cause wasting only in the flexors of the hip and extensors of the knee, with loss of the knee-jerk and anæsthesia in the front of the thigh, while the muscles below the knee present simple palsy with myotatic excess. Thus a man, who had had syphilis, complained of pain in the left groin and thigh, and presented wasting of the extensors of the knee, with anæsthesia in the front of the thigh; there was loss of the left knee-jerk, and a foot-clonus on each side. He recovered, but there was doubtless a small syphiloma at the upper part of the lumbar enlargement on the left side, interrupting the reflex arc, damaging the motor and sensory nerves, and compressing both pyramidal tracts. A growth in the membranes adjacent to the middle or lower part of the lumbar enlargement usually causes symptoms of wide distribution, affecting the whole of both legs, and the early symptoms of irritation often involve the distribution of the upper lumbar nerves. But a growth at the same level within the cord may affect chiefly the muscles of the lower leg, and cause anæsthesia in the region supplied from the sacral plexus. There may also be palsy and wasting of all the muscles of both legs from an extensive secondary myelitis, but even then the sensory loss is usually limited. In lumbar tumours there is early palsy of the bladder and sphincters. Examination of the sphincter and shows absolute paralysis, and not the pure reflex action present when the disease, arresting voluntary influences, is higher up the cord. The tendency to bedsores is commonly strong in tumours of this part.

Growths situated in the cauda equina, which are below and do not affect the cord itself, produce symptoms very similar to those due to tumours of the lumbar enlargement, but the symptoms are bilateral from the first in the majority of cases. Rarely one leg is affected alone

at the outset. The symptoms usually begin in the lowest parts of the legs, which are throughout affected in greater degree than the upper parts, and the flexors of the hip may escape altogether. Muscular contraction is much less common than in tumours situated higher up. The anæsthesia is chiefly in the region supplied by the sacral plexus, and the pains, usually very severe, are first felt in the same region, and there may be severe pain on each side of the sacrum radiating to the region of the sciatic nerves. The muscles below the knee rapidly waste, and often (but not always) present the degenerative reaction. These symptoms were well marked and characteristic in the case figured and described at p. 420 (Fig. 160), and the condition of the muscles suggests that the absence of any voltaic irritability in the muscular fibres was due to their early and intense fatty degeneration. In many cases the symptoms are irregular in distribution; the nerves are included in the growth, and are often damaged unequally, and less than might be expected from the size of the tumour, which may be considerable before its expansion is arrested by contact with the bony walls of the canal. Hence the loss of sensation may be partial, and reflex action may not be completely abolished. In the case above mentioned, for instance, a prick on the sole caused a reflex contraction of the muscles in the upper parts of the legs, but of these alone. In some cases, however, the loss of sensation has been absolute and reflex action also has been entirely lost. The tendency to trophic changes is great, and an affection of the sphincters is almost invariable. Cystitis and its consequences readily occur and may reach a high degree, in consequence of residual accumulation, when the patient is conscious only of slight difficulty of micturition.

Multiple tumours may give rise to very complex symptoms. If there are only two tumours they may be manifested by the successive development of focal symptoms, similar in character but different in seat. In many cases in which there are many growths only one or two of the largest have caused symptoms, and the smaller tumours have run a latent course. In some other cases in which many tumours have caused slight symptoms, these have simulated closely those of a system-disease of the cord. Thus, in the remarkable case recorded by Hughes Bennett, multiple growths on the posterior nerve-roots produced the characteristic manifestations of locomotor ataxy.* When, as has often been the case, cerebral tumours coexist with multiple spinal growths, the symptoms of the latter have usually been lost in those of the intracranial disease, which has preceded the spinal lesion.

The duration of the symptoms of tumours of the cord and membranes, those of syphilitic nature being excluded, varies from three months to five, or even ten, years. In the majority of cases death ensues at the end of from one to three years from the commencement of the symptoms.

* 'Clin. Trans.,' 1835.

DIAGNOSIS.—Although no symptom produced by tumours within the vertebral canal is actually pathognomonic, and the disease is of course not to be directly recognised, yet the combination of symptoms, and the course which they present, are in most cases sufficiently characteristic to render the diagnosis a matter of no great difficulty. The symptoms of greatest diagnostic importance are, first the pain, severe in degree and constant in seat, and secondly the progressive paralysis. Of less but still considerable importance are the rigidity of the muscles of the spine, the muscular contractions in the limbs, and the early and marked excess of reflex action when the cord itself is involved. In the course of the disease, the most important features are the steady progress of the symptoms, and especially their commencement on one side and extension to the other. Causal indications occasionally corroborate the diagnosis, and sometimes give additional weight to suggestive symptoms. Of such indications the most important are a history of constitutional syphilis, the presence of tubercular disease elsewhere, and, in rare instances, the existence, in other parts, of such multiple growths as are known to occur also within the spinal canal, especially multiple sarcomata and neuromata.

The diagnosis of multiple growths depends on the recognition of the successive development of the characteristic symptoms in more than one focus. It is rare for more than two of many tumours to cause definite symptoms. The greatest absolute difficulty in the diagnosis of a spinal tumour is presented by the cases, by no means rare, in which an intracranial tumour precedes the growth within the vertebral column, and the symptoms of the latter are overlooked in the presence of the severe manifestations of the former. In most cases, however, the disease can be recognised if the additional symptoms, which usually exist, are detected by careful observation, and their significance is recognised.

The differential diagnosis involves the distinction from diseases of the vertebral bones, of the membranes, and of the cord itself. Caries of the spine has many symptoms in common with tumour, but the root pains are rarely very severe, and the effects of compression of the cord are usually bilateral, either from the first or very soon after their commencement. The paralysis seldom reaches a considerable degree in one leg before the other suffers. The signs of bone disease are rarely long absent, and repeated examination usually reveals at least irregularity of the vertebral spines. From growths in the bones of the spinal column the diagnosis may at first be impossible. But the pain of tumour is rarely increased by movement to the same intense degree as when the growth commences in the bone, unless the tumour is in the cervical region of the cord, and then the greater accessibility of the vertebral column renders the exclusion of bone growths comparatively easy by local examination. In the dorsal region a growth in the bone may long escape detection, but even here the influence of movement on the pain is often extreme, far greater than

in the case of tumours within the canal. The effect of movement of the vertebræ on the nerve-roots, when compressed as they pass through the foramina, is far greater than when they are compressed within the canal. Sooner or later the local enlargement manifests itself externally and shows the nature of the case.

"Hypertrophic pachymeningitis," affecting the cervical region, is the only meningeal disease which closely simulates the symptoms of tumour, but its effects are usually from the first bilateral, and they have a considerable vertical extent. Hence it is only with central tumours, which grow through several inches or more of the cord, that the disease is likely to be confounded. In each malady there may be muscular atrophy in the arms, and paralysis, without wasting, in the legs. But in central tumours the sensory symptoms in the arms are usually much slighter than in the thickening of the membrane; there is rarely the early and limited anæsthesia which results from the damage to the nerve-roots in the latter disease.

The affection of the cord itself which most closely simulates the symptoms of tumour is chronic transverse myelitis. In such a case as that shown in Fig. 96, p. 240, the diagnosis may be a matter of extreme difficulty. The radiating pains in this case were severe and local, and the limitation of the symptoms, for a long time, to one side, with crossed sensory and motor palsy, presented an almost perfect analogy to the effects of a growth. But it is very rare for severe and acute radiating pains to result from focal myelitis, although a painful sense of constriction is common; moreover, in tumour, one half of the cord seldom suffers severely without the functions of the other half also being soon impaired in some degree. The limitation of chronic myelitis to a single focus is also very unusual; a considerable vertical extent of the cord is usually involved in varying degree. Hence the practical difficulty in diagnosis is not so great as, at first sight, might appear. Acute or subacute myelitis can only be confounded with a growth in cases in which inflammation results from pressure, and develops with independent energy and rapidity. In such cases the occurrence of myelitis must be recognised, and the diagnostic problem is the detection of the preceding symptoms of the growth that excites the inflammation. This is usually easy; the severe pains, and commonly also the slighter symptoms of paralysis, which existed before the rapid increase in the loss of power, are sufficient proof of a chronic morbid process, to which the inflammation is secondary.

The early pains are often thought to be neuralgic, but their constancy and continuance should suggest organic irritation; their seat moreover is rarely that in which neuralgia is common. The pain produced by tumours of the cauda equina is often felt first in the sciatic area, and thought to be sciatica, but it is commonly from the first bilateral, while sciatica is almost invariably one-sided, and bilateral sciatic pain should always suggest irritation where the sensory fibres from both sides are

near together, and can be irritated by a single cause, *i. e.* it should suggest disease within the vertebral canal. In all cases, however dubious the early pains may be, other symptoms soon become added, and prove that there is more than a simple neuralgia.

If the existence of a tumour is clear, the question remains, what is its nature? If the patient has had constitutional syphilis, the syphilitic nature of the growth is highly probable, and the probability is further raised in degree if the growth developed rapidly, so that the symptoms attained a considerable intensity in less than three months from their onset. The coexistence of a cerebral lesion also increases the likelihood of the syphilitic nature of the growth. Either of the two last considerations suggests the same conclusion in any case in which syphilis, although not proved, is possible, but it must be remembered that cerebral and spinal tumours, of other than syphilitic nature, may coexist. In the same way, tubercular and scrofulous processes suggest the tubercular nature of a tumour, and this is further supported by indications of the existence of more than one tumour in the spinal cord, and by long periods of arrest of the progress of symptoms that were slow in their development. Such arrest indicates an arrest of the growth of the tumour, which we cannot infer from the subsidence of symptoms that developed rapidly, and may have been due to secondary inflammation. Tumours elsewhere always constitute strong evidence of the nature of one in the cord, but such an indication is rarely available, since the spinal canal is rarely the seat of secondary growths. Multiple tumours are probably either sarcoma or neuroma. In the absence of these indications a meningeal tumour is probably a sarcoma, and a tumour within the cord is probably a glioma, because these are the most frequent kinds of growths which occur in those situations. A diagnosis founded on mere frequency of occurrence has necessarily only a low degree of probability, and must be sometimes wrong.

PROGNOSIS.—Unless the tumour is syphilitic the prognosis is necessarily most grave. Of all syphilitic lesions, growths are those that are most amenable to treatment, and the effects of which most certainly pass away, if they have not reached an extreme degree or lasted too long a time. If, however, the damage has been allowed to remain, unchecked, for several months, recovery may be imperfect. Tumours of other kinds steadily increase, and the resulting damage is on the whole steadily progressive although its course may be varied by stages of rapid (myelitic) increase in the symptoms, followed by stationary periods and even by transient improvement.

TREATMENT.—If the growth is syphilitic, iodide of potassium should be given in full doses of 30—90 grains a day, and unless the symptoms rapidly lessen, mercury may also be administered. It is important that the treatment should be energetic and prompt, so as to effect

some diminution of the pressure as quickly as possible, since the longer this continues, the more considerable are the degenerative changes. Even a difference of a few days in the influence of treatment, and consequent duration of pressure, may make a difference of weeks in the duration of symptoms and have a distinct influence on the degree of ultimate recovery. If the symptoms suggest a tumour, and syphilis is possible, appropriate treatment should be adopted for a time, since it can do no harm if the tumour is not syphilitic, and may save the patient's life and strength if it is. It should be remembered, moreover, that syphilis can only be excluded when there has been no *possibility* of infection. In a certain proportion of the cases of late syphilitic lesions, in the nervous system and elsewhere, there is no history of secondary syphilis, and in another proportion of cases of secondary syphilis there is no history of a primary sore. Hence it is *à priori* certain that in some cases of late lesions a history of both primary sore and secondary symptoms will be absent, and, as a matter of fact, such cases are met with not unfrequently. Of course these cases cannot be counted as syphilitic in any scientific investigation into the influence of this disorder, but we have often to allow weight, in treatment, to considerations which are insufficient to influence scientific investigations, and treatment determined by the mere possibility of syphilis is often justified by its results.

In other than syphilitic cases we can do little more than treat symptoms, relieving pain by sedatives, watching the state of the bladder, preventing the occurrence of cystitis and guarding the patient from bedsores. We must remember that sedatives will probably be needed for a long time, and they must therefore be used as sparingly as possible, lest custom and tolerance deprive them of their power. Cocain may be used in many cases as an aid in economising the influence of morphia. It is highly probable that Surgery may ultimately be able to cope, in some degree, with meningeal tumours. Modern methods render the opening of the spinal canal far less formidable than it formerly was, and the removal of a tumour from the membranes of the cord would involve less immediate danger of serious consequences than the removal of a tumour from the brain. On the other hand, growths within the spinal cord are altogether outside the possible range of surgical procedure, since the removal of such a tumour would entail an injury equal to the greatest damage the growth could ultimately inflict, and equally disastrous in its consequences.

CAVITIES AND FISSURES IN THE SPINAL CORD

(SYRINGOMYELIA, HYDROMYELIA, HYDRORRHACHIS INTERNA).

Cavities in the spinal cord, distended with liquid, are met with at all ages. They are found sometimes in young children, a condition termed "hydromyelia." In such cases they are clearly due to an arrest of development, which often affects the brain also. The cavities met with in adults have been distinguished from those found in young children, and have been termed "syringomyelia." This condition has been supposed to be acquired, the result of morbid processes commencing in adult life, but the two conditions present so many points of resemblance that they are probably of the same nature, and the adult cases, as well as the infantile forms, are congenital in origin, although they are no doubt modified by secondary changes occurring during their longer course.* The following figures illustrate all the most important facts regarding this morbid state, and the somewhat complex conditions will be best understood by considering the appearances presented in the several figures, and their significance.

The great fact of all cavities within the cord is their association with the presence of abnormal tissue, resembling closely that which commonly surrounds the central canal of the cord, and that which exists as a thin layer on the surface of the cord, between the pia mater and the nerve-tissue. The two are probably of the same nature, but that around the canal is modified by the growth in it of small cells or nuclei. It is probable that this abnormal tissue, accompanying the cavities, is due to a persistence of the embryonal tissue from which the cord is developed, and that the presence of the cavities is due partly to defective closure of the tube which is first formed (residual portions of which become distended by accumulation of liquid), and partly to the breaking down of this persistent embryonal tissue or to tissue formed from it by a process of growth. The cord sometimes suffers serious damage from pressure, owing to the distension of the cavity by the liquid which accumulates within it.

To understand the origin of these conditions it is necessary to remember the mode of development of the spinal cord. The sides of the primitive furrow of the embryo coalesce so as to form a canal of relatively large size, the walls of which are thinner in front and behind than at the sides, and consist of elongated cells, some of which are arranged regularly at the inner surface of the cavity, like an epithelium. The anterior wall first becomes thickened, to form the white commissure and the front part of the grey, while an abundant growth of cells occurs at the sides of the tube, and extends forwards (*i. e.* downwards in the position of the embryo), thus forming the

* See Leyden, 'Virchow's Archiv,' Bd. 68, p. 1.

lateral and anterior columns; between the two projections thus formed is the depression of the anterior fissure. Next occurs a growth backwards of the now thick sides, which forms the posterior parts of the lateral columns and the adjacent part of the posterior columns, and with this the posterior roots are connected; this forms the "root-zone" of the posterior columns. The posterior median columns are formed later. The central canal extends backwards between these two rudimentary postero-external columns, being closed behind only

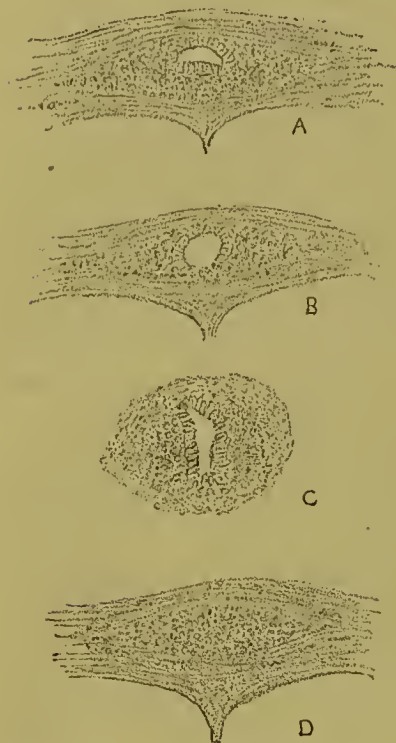


FIG. 164.—Central canal in normal cords. It has the form of a transverse slit in A, a vertical slit in C (from the conus medullaris), while in B it is circular. In each it is lined by columnar epithelium and surrounded by cellular elements, rounded, or angular from compression, mingled with granules. In D, which is from the same spinal cord as C, the position of the canal is occupied by a mass of nuclear tissue.

by a thin layer of cells. As the posterior columns increase in thickness the posterior part of the canal becomes narrowed, and its walls unite near its anterior extremity so as to form the posterior grey commissure, and divide the canal into two parts. Of these the anterior becomes the permanent canal, while the posterior is reduced to a narrow fissure between the posterior columns. Ultimately this posterior narrow part becomes closed by a growth of cells which occurs from behind forwards, as the median parts of the posterior columns develop. All these parts consist first of embryonal cells, which afterwards undergo a transformation into the nerve-elements proper, and the order of transformation is the same as that in which the parts were formed, the posterior median columns latest. The cells of the grey matter are formed before the fibres of the white columns. Some of the embryonal elements undergo a different and slighter change. They may almost be said to persist, constituting the neuroglia and the gelatinous grey substance continuous with it around the cord, at the posterior root fissure, and around the central canal.

The central canal ultimately lies in the anterior part or middle of the grey commissure. It may persist through life as a cavity, circular in section, or as a slit, antero-posterior or transverse, and is lined with epithelium (Fig. 164). Around the canal there are usually many nuclear elements, especially on each side of it, and these frequently fill up the lumen of the canal, so that its position is indicated only by an oval mass of small cellular elements (Fig. 164). The obliteration of the canal often occurs quite early in life, and is no evidence of any morbid process. The canal may be found obliterated

in one part of the cord, and patent in another (c, d, Fig. 164). The simple closure of the canal by nuclei does not cause any distension of the part which is above the closed portion, or, at any rate, not more than a very slight increase in size, so trifling that its significance is open to question.

In the congenital condition found in young children the morbid change presents two distinct features. There is usually a quantity of tissue of translucent aspect, which, from its structure, is clearly persistent embryonal tissue in which nerve-elements have not developed, and there is a cavity which is usually clearly due to imperfect closure of the canal. The change is always confined to the posterior half of the cord, and usually to the posterior columns, which, as we have seen, are formed last. The persistent embryonal tissue is sometimes increased by a process of active growth, and the persistent canal or cavity is often enlarged by a process of distension. Further, when the persistent tissue is considerable in quantity it may break



FIG. 165.—Hydromyelia, from a child two years old, with encephalocele and absence of cerebellum. (After Leyden.) In A, cervical region, the central canal is large, lined with epithelium, and from it a medial fissure extends backwards nearly to the periphery of the cord, limited by a layer of homogeneous tissue, more abundant at the posterior limit. In B, lower down, the fissure is enlarged to a cavity of considerable size, while in C it is still larger, and the posterior columns are reduced to a narrow zone between the layer of tissue which bounds the cavity and the grey substance.

down, and thus give rise to a new cavity, or enlarge that which has developed from the primitive canal.

These conditions are shown very well in the adjoining figures, after Leyden (Figs. 165, 166). In the former, at A, the cavity presents nearly the shape which it has at one period of development, before the formation of the posterior columns is completed, and without the separation into two parts produced by the formation of the posterior commissure. It is bounded by a narrow layer of embryonal tissue, which widens out behind, having nearly the shape of the posterior median columns, which are, as we have seen, the last to be formed. In B and C the cavity is much larger, and the posterior columns are smaller, either by an earlier arrest of development or by greater distension, or, most probably, by the influence of both these agencies. The cavity extends up to the posterior surface of the cord, and is closed in there only by a narrow layer of cellular structure.

In Fig. 166 we have somewhat more complex conditions. The cavity in the first section closely resembles in appearance that in A of the last figure, but differs in the important fact that the posterior com-

missure has apparently been formed and the central canal developed in the normal manner, although it has become obliterated by nuclei.



FIG. 166.—Hydromyelia, from a child aged two and a half years, with internal hydrocephalus and absence of cerebellum. (After Leyden 'Virchow's Arch.,' Bd. 68.) A, B, cervical region; gelatinous (embryonal) tissue in the posterior columns encloses a medial cavity, lined in places with cylindrical epithelium, and believed therefore to be the dilated central canal. C, lower dorsal; in each posterior column is a mass of similar gelatinous substance (which, by a mistake, is shown in C as an open cavity). In D, lumbar region, this tissue occupies only the position of the posterior median columns.

This is clear if the commissure is compared in the several figures. The cavity therefore does not, as in Fig. 165, represent the whole of the primitive canal, but only the posterior portion, after its division. The adjacent tissue is much more abundant than in the other case. The projection into the posterior extremity of the cavity indicates a process of active growth, which is also shown by the irregularity of the cavity in B, the large amount of tissue, and the wide separation of the posterior horns. In C, from the lower part of the cord, the embryonal tissue occupies two oval areas in the posterior column (clear in the figure), while the cavity is closed or nearly so. (A little lower down a cavity existed in the centre of these areas, apparently due to breaking down of the tissue.) In D, again, the embryonal tissue occupies only the middle line, as a wedge-shaped area, due to the defective formation of the medial part of the posterior columns. It has been mentioned that the neuroglia may be regarded as a persistent, slightly modified, embryonal tissue, and these exuberant masses of tissue bear considerable resemblance in structure to glioma, so that the condition has been called "gliomatosis." In each of these cases the brain participated in the defect of development; there was internal hydrocephalus, and the cerebellum was absent.

The cavities in the adult cord present many varieties. The most common form resembles in its essential features the congenital disease which we have just considered. Between the two there are also differences, but the resemblance is sufficiently close, and the origin of the differences is sufficiently clear, to make it practically certain, as already mentioned, that the two forms are really the same, and the condition met with in adults is the congenital condition persisting until later life. Of the characters which this form has in common with the congenital disease, the most important are the posi-

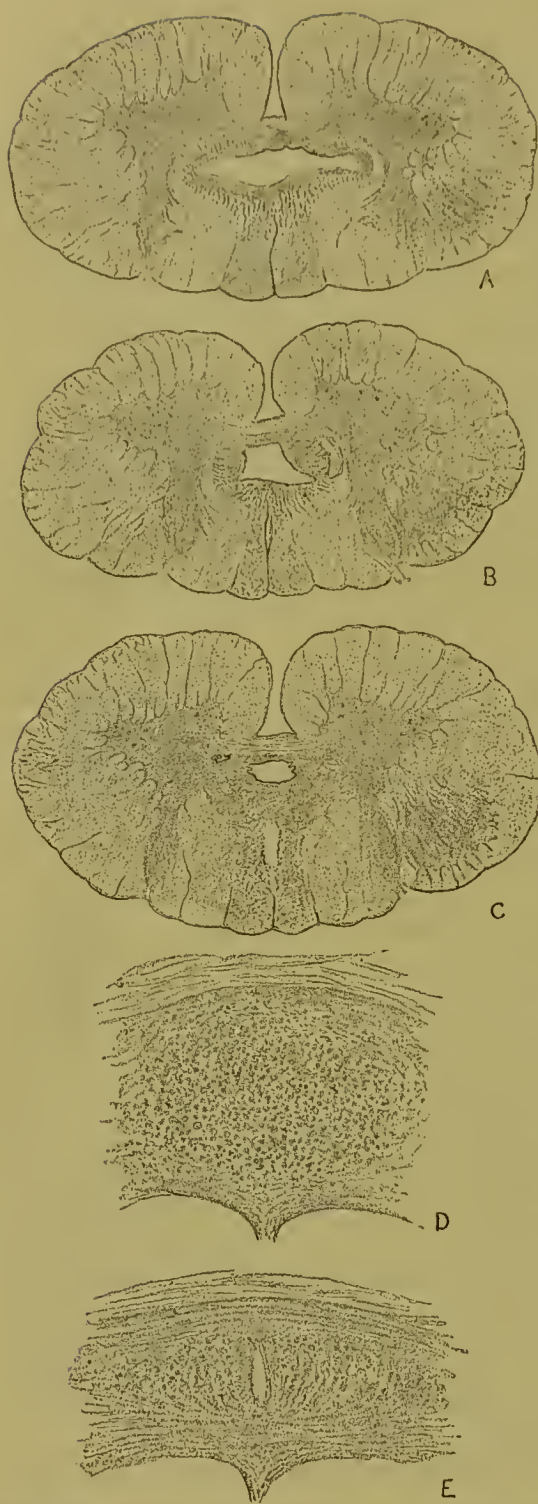
tion of the cavities, and the presence of tissue in their vicinity of embryonal or gliomatous nature. The differences depend on an apparent increase of this tissue by a process of growth, on the greater damage to the other parts of the cord by the distension of the cavity, and on the extent to which the formation of new cavities or enlargement of old ones takes place by a process of disintegration of the newly-formed or persistent tissue.

In the first place, as the simplest condition, we may have a dilatation of the central canal surrounded by gliomatous tissue disposed as in congenital cases. This tissue is often more abundant, and forms a more distinct mass, lower down the cord, below the cavity. A good example of this condition in slight degree is presented by the cord, the seat of chronic myelitis, represented at Fig. 97, p. 241. In the cervical region (A) there is a large central canal, bounded by a thick layer of gliomatous tissue. In the inner layer of this tissue, and bounding the cavity, is a sinuous membrane, fibrous in structure. The origin of this membrane is not easy to explain, but it is often found in cavities of this character, and is evidence of the common nature of those that differ much in other respects. In the lumbar region (B), the tissue, which bounds the cavity in the other section, forms a large round mass in the position of the canal, obliterating it.

In other cases a central cavity appears not to be the canal itself, but to be situated behind it, as we have seen may be the case in infants. It is then apparently due to a persistence of the fore part of the posterior portion of the canal, after the posterior commissure has been formed. In Fig. 167, for instance, we have a central cavity, but in the commissure in front of this is a mass of nuclei having the usual aspect of an obliterated canal.* The cavity is surrounded by a zone of tissue, which in A and B sends off a fringe of processes, especially backwards. This tissue increases in the lower part of the cervical region (C). In B a small cavity is formed between the tissue and the grey matter, by breaking down, and in C a small cavity exists in the middle line behind the growth, probably due to deficient closure of the original fissure, and increased by a destructive process. Still lower the commissure resumes its normal appearance, but the collection of nuclei in the position of the canal is unusually large (D). In the lumbar region the canal is patent (E). The degenerations of the posterior median column and of the lateral columns were secondary.

We have seen that even in the congenital cases we must recognise

* This is the probable interpretation. At the same time it is possible that the nuclei have not this significance, and that it is really the central canal. It is often more difficult than might be imagined to say whether a cavity does or does not represent the central canal. The presence or absence of an epithelial lining has generally been taken as a criterion, but it is doubtful whether this has any significance. Around the whole of the original cavity the inner layer of cells is arranged as an epithelium, and if any part of it persists it is probable that epithelium will persist also. Moreover, the epithelium often disappears from the wall of the dilated central canal itself; always where it is enlarged by breaking down of tissue.



a process of growth of the persistent embryonal tissue to account for some of the conditions met with, and a like process is suggested by the central mass in Fig. 97, B. Hence it is not surprising that in many cases the growth should attain the dimensions, and assume the characters, of a positive tumour. In most cases the tumour has been central in position, and has had the structure of a glioma. It has occupied a large part of the area of the cord at a certain part.* In the chapter on the tumours of the cord it has been pointed out that sarcomata also may grow from the tissue around the central canal, as in the case shown in Fig. 162. Hence it is not surprising that the condition of syringomyelia, even if this is congenital in origin, should be frequently associated with definite tumours. The growths have often been multiple, apparently the result of a widespread tendency, and,

* As in an interesting case described and figured by Riesinger, 'Virchow's Archiv,' Bd. 98.

FIG. 167.—Syringomyelia, from a case of tumours of the pons and cauda equina, with some diffuse sclerosis in the dorsal region of the cord. A, B, and C are sections of the cervical enlargement. The large cavity in A is perhaps the dilated canal, since it is lined in

front with epithelium, but in front of it, in the posterior commissure, is a group of nuclei like an obliterated canal. The zone of tissue around the cyst is composed of interlacing fibres and nuclei, and processes extend from it into the posterior columns, both in front and behind. In B the cavity is smaller and the wall thicker, while in C the tissue about it forms a considerable mass, and a second small cavity has formed in the middle line, apparently by the breaking down of the tissue. In the dorsal region there was merely an unusually large accumulation of nuclei in the position of the canal, which, in the lumbar region, had the normal aspect (E). The cord presents degeneration of the right pyramidal tract (descending from the tumour in the pons), and of the post. med. cols. and ant. lat. ascending tract, probably ascending from the sclerosis.

perhaps, connected with a widespread persistence of embryonal tissue, of which we shall presently consider a remarkable instance. In the case shown in Fig. 167 there was a tumour of the pons and also one of the cauda equina. The nature of the growths is uncertain, but they were probably either sarcoma or glioma. The same tendency to morbid growths is illustrated also by Fig. 168, which is similar in many respects to that just considered. In this case also there was a tumour of the pons and one of the cauda equina, and there was also a central growth in the dorsal region, occupying the greater part of the area of the cord, to which the ascending degenerations are secondary. In A there is a large central cavity which is probably not the central canal, since, as in the last figure, the position of this is marked by the oval group of nuclei in the anterior part of the grey commissure. The

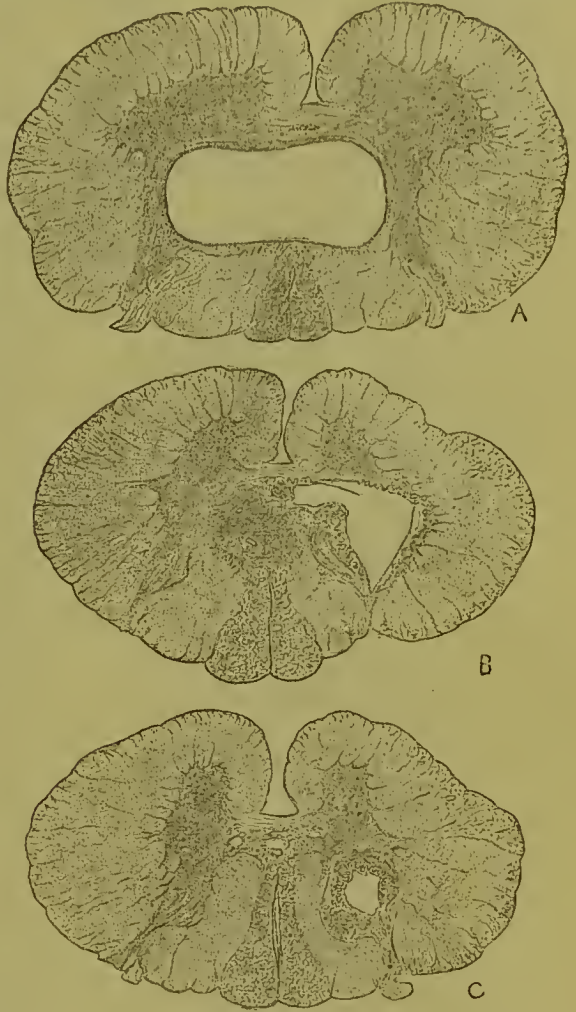
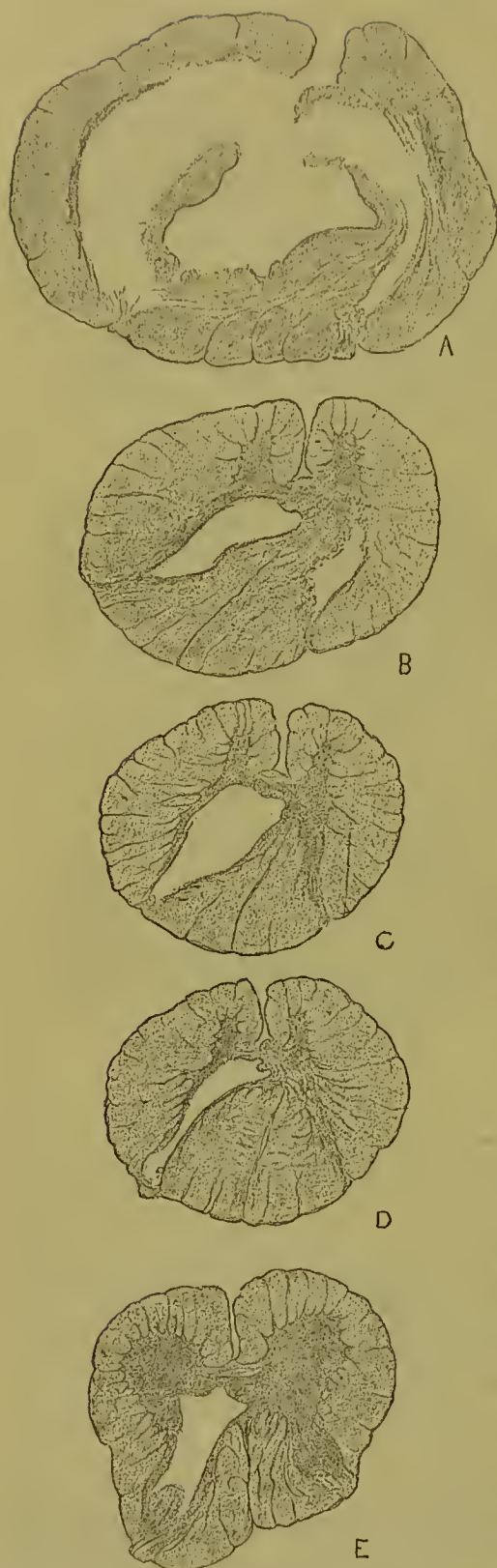


FIG. 168.—Syringomyelia, from a case in which there were tumours in the dorsal region of the cord, the cauda equina, and the pons Varolii. A, mid-cervical region. A large oval cavity lies behind the grey commissure, surrounded by a narrow wall, chiefly composed of fine fibres, but with a wavy membrane on the inner surface. In the front of the posterior commissure an oval group of nuclei has the aspect of an obliterated central canal. A few short processes extend from the wall of the cavity into the posterior columns. There is secondary ascending degeneration of the post. med. cols., right direct pyramidal tract, and ascending antero-lateral tract in all the sections. B, first dorsal. Behind the posterior commissure is a growth consisting of small cells, round and fusiform, the latter with round nuclei. It contains many vessels with thickened walls. A fringe of fine processes extends from it into the posterior columns. An irregular cavity occupies, on the right side, the posterior half of the grey matter, which is reduced to a narrow layer around the cyst. This is lined by a delicate nucleated membrane, outside which is a thicker membrane, lying in folds, the section of which is thus sinuous. Outside this again is a small-celled growth similar to that behind the commissure. In C, a little lower, this cavity is smaller, the sinuities of the wall greater, and the growth outside it more abundant. (Still lower the area was entirely occupied by the growth.) In the posterior commissure there is only a quantity of loose nucleated tissue in the situation of the growth.*

* For the opportunity of drawing these sections I am indebted to Dr. Dreschfeld. A full account of the case has been published by Dr. Harris in 'Brain,' Jan., 1886. The large growths were sarcomata.



zone of tissue which bounds it is narrow, but a few processes are given off behind. In B, first dorsal, this tissue forms a compact mass, and the processes are numerous and very similar to those in the last figure. In C, the commissure is reduced almost to normal conditions. In this cord we have also an instance of the fact that cavities may occur in other parts, very similar in their general characters to those that occur in the central region. In B, a large cavity occupies the intermediate grey matter and posterior cornu on the right side, with some morbid tissue bounding it on the medial side. In C, the cavity is smaller and the tissue around it is more abundant, but easily distinguishable from the grey substance, while a little lower down the cavity disappeared and the tissue formed a compact rounded mass. The cavity is lined by a delicate layer of cellular membrane; outside this is a fibrous membrane, lying in folds which appear as sinuosities in the section. The existence of this membrane shows that the cavity is not simply formed by the breaking down of the tissue. The tissue is quite similar in structure to that which lies behind the commissure in B.*

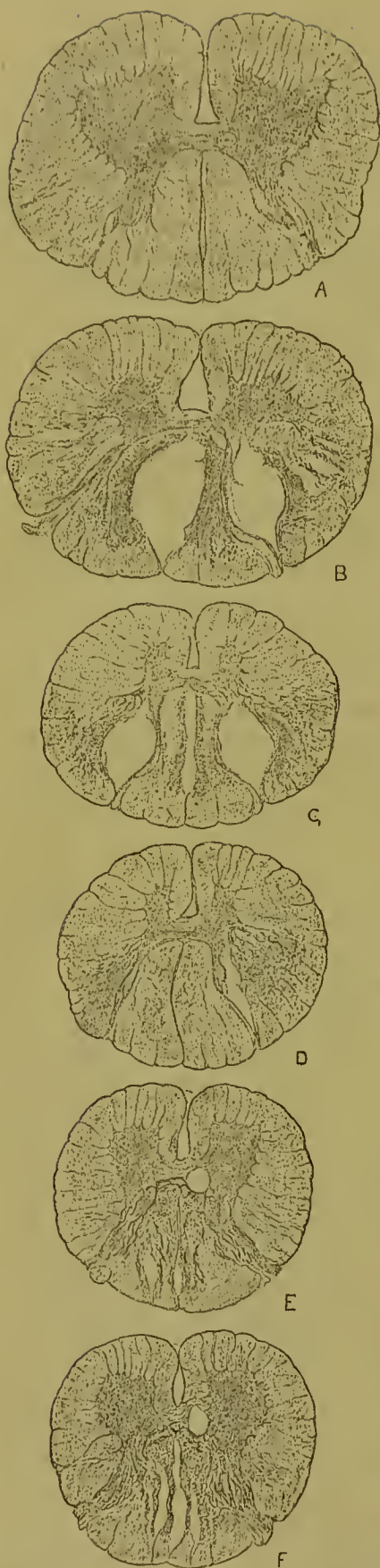
* In connection with the tumour of the cauda equina in these two cases, it may be mentioned that the same coincidence has been observed in other

FIG. 169.—Sections of a spinal cord in which a cavity existed throughout its length. Hæmorrhage in the cervical region was the immediate cause of death. The extravasation had there distended the cord (A), and had burst into the cavity, which was filled with blood. A smaller cavity existed in the right cornu, in the upper dorsal region (B). In most parts the inner surface was slightly irregular, as if produced by breaking down of the tissue, but near the position of the central canal a sinuous membrane existed in places, without, however, any epithelial covering.

The posterior cornu is not an uncommon seat of cavities, which may extend through the whole length of the horn. An example of this is shown in Fig. 169. Hæmorrhage had occurred into the cervical region of the cord (A), which was enlarged and distended with blood, so that the precise character of the cavity in this region was indistinguishable; the blood also filled the cavity through the entire length of the cord. In the upper dorsal region (B) there are two cavities, one in each posterior cornu, but that on the right ceased a little lower down, while the other extended to the lumbar region, as shown in the figure, reaching almost to the surface of the cord. The upper and inner extremity of this cavity is in the position of the central canal, no other trace of which exists. There is some abnormal tissue in the neighbourhood of the commissure, and also in C, irregular tracts pass transversely from the median septum in the fore part of the posterior columns. The presence of this tissue, and the relation to the central canal, suggests a congenital origin. During the reduction in size of the canal, in the development of the cord, the cavity presents at one period short lateral processes (compare Fig. 171, A), and we can therefore understand how a still earlier arrest of development may lead to the persistence of a lateral process on one side or both, surrounded by persistent embryonal tissue, and that the cavity should afterwards extend in this tissue or in the grey matter by a process of disintegration compare Fig. 171, D. A comparison with this figure (E) suggests that the two cavities were united in the middle line in the upper part of the cord, in which the hæmorrhage took place.

In the conditions we have hitherto considered, definite limited regions of the cord have been involved through a considerable vertical extent. The changes are sometimes, however, much more diffuse in their distribution, as is shown by the remarkable lesions represented in Fig. 170. In this cord, which is that of an adult, we have a combination of the same two conditions of abnormal tissue and cavities. Scattered through the nerve-substance are tracts of a peculiar tissue represented by the darker shading of the figure, and in many regions thus affected there are cavities, for the most part fissure like, as if the cord had been split here and there. At first sight the appearance suggests an active and recent morbid process; nevertheless, I think that the congenital origin of the condition is beyond doubt. It will be observed that the morbid changes, like the other forms of syringomyelia, are chiefly in the posterior half of the cord; small areas of disease in the anterior columns, in the sections E and F, are the only exceptions. The minute structure of the abnormal tissue is exactly like that of the normal gelatinous layer beneath the pia mater. Besides the conspicuous areas figured, it could be traced here and there, in small tracts, through the

eases. There was a tumour in that situation, for instance, in the case described by Riesinger, who, rightly regarding the condition as congenital in origin, suggests that the tumour may be connected with the fact that the embryonal cord occupied the entire length of the canal.



whole of the posterior columns and the hinder parts of the lateral columns, and also in the posterior horns, in the grey matter of which it could be readily distinguished. The relation of the cavities to this substance is distinct; where they extend beyond it this is apparently the result of the distension of the cavities and an actual fissuring of the cord, along the lines of connective tissue, by the pressure of the effused liquid. The medial cavity in c is doubtless due to defective closure of the original fissure between the posterior columns. In the lumbar region the nerve-fibres are absent from the front of the posterior columns, where a coarse network of this residual tissue encloses empty spaces.

SYMPTOMS.—In many cases syringomyelia is discovered after death when it has given rise to no symptoms and its existence has not been suspected during life. In young children it appears never to cause any definite symptoms. In adults, however, considerable disturbance of function is sometimes

FIG. 170.—Cavities in the spinal cord. A, mid-cervical; presenting merely some increase of connective tissue in the left lateral column. In B there are several cavities having the aspect of distended fissures, the largest in the middle line of the posterior columns and the right posterior cornu; smaller ones in the lateral columns. Adjacent to each is a quantity of dense tissue, which had a peculiar and uniform structure. It consisted of a very fine network of fibres with minute nuclear bodies in the interstices. No larger cells could be seen. Throughout the posterior and lateral columns this tissue could be seen, here and there, thickening the neuroglia, where no conspicuous masses were formed. It could also be traced in the posterior cornu, and was readily distinguished from the spongy substance by its slighter staining. The distribution of this tissue is shown in the other sections by the darker shading. In C the fissure in the middle line is narrow. In E and F the anterior part of the posterior columns contained a coarse network of this tissue, and no nerve-fibres. The central canal was obliterated, but the group of nuclei that marked its position could be traced throughout the cord.

produced. In some cases the growths, which often coexist and which probably start from the residual tissue, may cause the symptoms of tumour. These are often not characteristic, on account of the central position of the growth; the irritation of the nerve-roots is commonly absent. In other cases the distension of a central cavity may cause such compression of the white columns and grey matter as leads to definite disturbance of function.

Such disturbance generally involves the central functions of the cervical enlargement of the cord, because it is in this region that the cavities attain their largest size, and the nerve-elements of the cord suffer in greatest degree. The grey matter is often especially damaged by pressure, and hence muscular atrophy is one of the most conspicuous of the symptoms that are produced. With it is usually associated loss of sensibility, due to the damage to the posterior grey matter and nerve-roots. The wasting resembles in its characters that of progressive muscular atrophy; the muscles of the shoulders and back have suffered more frequently than those of the forearm and hand. The cutaneous anæsthesia usually involves the same region as the muscular wasting, but extends beyond the limits of the latter, and may involve, for instance, the forearms and hands, while the atrophy is limited to the shoulders and upper arms. Pain is not usually a prominent symptom. The conducting functions of the cord are also involved in some degree, but often much less than the central functions, so that there is merely slight weakness of the legs when there is considerable atrophy in the arms. In other cases, however, the paralysis of the legs has been considerable, and there have been muscular contractions in the unwasted parts. The sphincters are often involved, and trophic changes in the skin are frequent, and occasionally are severe. When disturbance of the central functions has developed, the disease usually runs a progressive course and terminates in death at the end of two or three years.

Even when well-marked symptoms have been present, they have so far resembled those of other more common maladies, that the lesion has been seldom suspected during life. The combination of atrophy with more extensive anæsthesia, in the absence of considerable pain, would justify a suspicion of the disease. Pachymeningitis, which also causes muscular wasting and sensory loss, is usually attended with a considerable amount of pain in the affected region, and the anæsthesia is not more extensive, and is usually less extensive, than the muscular wasting. It is not likely that any treatment can influence the morbid process.

SPINA BIFIDA.

Spina Bifida depends on a defect in the closure of the vertebral arches. It is most common in the lumbar region of the spine; sometimes is present at more than one place, very rarely in the whole length

of the vertebral column. It is present in about one child out of every thousand born (Chaussier). The spinal cord is sometimes normal, although the fluid outside it is greatly increased in quantity, and distends the external sac. The cord, however, generally reaches to the lower extremity of the vertebral canal, as it does at an early period of development, being kept adherent at its lower extremity by the morbid process. In many cases the central canal of the cord is enlarged, and the lower part of the cord may be distended in what has been termed "internal hydro-rhachis." In this condition the central cavity may be closed in behind by a very thin layer of nerve-substance. More frequently, however, the cord is the seat of a developmental defect similar to that of the bony canal. In the lumbar region the primitive canal has failed to close, so that the cord is open posteriorly, and the two posterior columns may even be far apart, so that the cord has the form of a thick lamina in the anterior wall of the sac.

An example of this condition is shown in Fig. 171, which illustrates also another fact of the disease, viz. that some developmental defect is often present in other parts of the cord. In the cervical region the grey commissure is unusually large, and the canal is cruciform, a shape which it presents at one period of development; and which is here persistent. This condition obtains throughout the dorsal region until, in the lower part, the tissue behind the commissure is so abundant as to unite the necks of the posterior horns. Below this the canal enlarges into a condition of syringomyelia, surrounded by the abnormal tissue, which, as we have seen, is constantly associated with enlargement; the canal is limited, moreover, by the sinuous membrane that is so often met with. The cavity extends also into the posterior horns, clearly by a process of disintegration, since in D the membrane lining the enlarged canal persists and marks the limit of the canal and the extent of the simple disintegration. The progressive separation of the posterior columns is shown in F, G, and H. In the lowest part

FIG. 171.—Spina bifida: sections of a spinal cord. In A, cervical region, the only abnormality is a large cruciform central canal and an unusually deep grey commissure. This continued through the dorsal region, at the lowest part of which a great change occurred. In B the canal is larger, and the necks of the posterior horns (and posterior vesicular columns) are united by much commissural tissue; in this many vertical fibres could be seen, and fibres ran from before backwards in the middle line. In C an extension of the canal backwards has taken place, and it is bounded by a sinuous membrane. The outer part of the grey substance is atrophied. In D the cavity has extended into each posterior horn almost up to the surface of the cord, in part by a breaking down of tissue, because the membrane limiting the enlarged canal remains undestroyed. In E, still at the junction of the dorsal and lumbar region, the cavity has receded from the left horn. In F the division of the cord has taken place, not in the direction of the cavity in the horn (which is filled up), but in the middle line, at, or close to the median septum. In G a wider separation of the posterior columns has taken place, and a cavity has formed on the right side, which extends into the horn, and almost separates the posterior column. In H the cord is spread out and formed part of the wall of the sac. The posterior nerve-roots (*p r*) mark the position at which the posterior horn comes to the surface. The grey matter is in many parts atrophied and translucent, and in H contains many large vessels. The septal lobulation on the surface of the cord is greater than normal.

FIG. 171.



of the cord the development of the posterior columns has been apparently hindered, as will be observed if *a* be compared with *f*. In the former there are also some fissures in the grey substance, also formed apparently by disintegration. The grey substance of the cord is conspicuously wasted in the lumbar region, thin and translucent.

In cases of spina bifida in which the cord is normal there are commonly no symptoms. When the central cavity is large, and the lower part of the cord is greatly distended, or when, as in the case figured, the cord shares the defect of union, there is frequently paralysis of the legs and sphincters, often with muscular atrophy, which is explained by the wasting of the grey matter. Occasionally the child has been unable to stand, although the legs have been neither wasted nor weak; perhaps the inability in this latter case has been due to the arrested development of the posterior columns.

The treatment of spina bifida is purely surgical.

TRAUMATIC LESIONS OF THE SPINAL CORD.

CAUSES.—In fractures and dislocations of the spine, the cord generally suffers compression or laceration, as already described in the chapter on injuries of the vertebral column. The cord may also be directly wounded by stabs and gunshot injuries, in which it may be divided, completely or partially. Such injuries furnish a considerable proportion of the cases of unilateral lesion of the cord. In gunshot wounds the cord more often suffers from displaced fragments of bone than from the ball itself.

Far more common than direct injury is damage to the cord by concussion of the spine, either local or general. Gunshot wounds occasionally furnish examples of local concussion; a bullet may strike the spinal column, and lodge in its vicinity, with the effect of causing immediate paraplegia, as complete as if the cord were divided, and yet it may be found that the spinal column has not been injured, and the cord is merely softened at the spot. Other causes of concussion are the fall of heavy bodies on the back, such as a beam of timber or a sack of corn, a blow on the back from some blunt weapon, a fall upon the back, either on a flat surface or on some projecting object. Less frequently the cord suffers from a general concussion of the body, in which the spinal column is not specially involved. The cervical region is occasionally damaged in falls on the head. Railway accidents are frequent causes of concussion of the spinal cord; the back being struck with violence when the body is thrown from one side of the carriage to the other. Another common cause is a fall from a horse upon the back, or a fall downstairs, in which the spinal

column is bumped against the edges of the steps. Occasionally effects very similar to those which are produced by a blow on the spine, are caused by a sudden contraction of the spinal muscles in some violent effort, a "rick of the back" as it is popularly termed. It is possible that, in such cases, the primary damage is sometimes to the vertebral ligaments and articulations, and that the cord suffers secondarily.

PATHOLOGY.—The anatomical lesions, in cases in which the vertebral column is not injured, vary much in different cases. Hæmorrhage is often found, sometimes outside the dura mater, sometimes on the inner surface of the membrane, in the pia mater, or in the substance of the cord itself. Occasionally the substance of the cord has been found lacerated when the vertebral column has not been injured. In many cases there is local softening, commonly yellow in tint, sometimes mingled with red, often involving the whole thickness of the cord, and occasionally extending, as central softening, through a considerable vertical extent. Under the microscope, the usual products of degeneration are seen, sometimes with hæmatoidin crystals. Such softening may occur rapidly in severe local concussion, and be found complete a few weeks, or even a few days after the injury.* On the other hand, in some cases of complete paralysis, no lesion of the cord has been found, either with the naked eye or the microscope, a few days after the injury.† In other instances, in which the cord is examined some weeks or months after the accident, the signs of chronic myelitis are found, in scattered foci or more diffuse tracts, in the white columns or grey substance. The nerve-fibres are wasted, and the connective-tissue elements are increased in quantity, and, in the early stages, there may be a leucocytal infiltration about the vessels, dilatation of the capillaries, and often minute extravasations, although none were visible to the naked eye. In the anterior cornua, the motor nerve-cells may be damaged, sometimes swollen and vacuolated, or shrunken, and the anterior root-fibres may be degenerated. The grey matter is especially apt to suffer when the enlargements are injured; in the dorsal region the change may be confined to the white columns. The usual ascending and descending degenerations may be found above and below the most damaged parts. Occasionally there are indications of meningitis, diffuse or disseminated, and sometimes confined to the dura mater.

SYMPTOMS.—The effects, immediate and remote, of injuries of the spinal cord extend over almost the whole range of symptoms of cord disease, and their variations in character and course are almost infinite.

* Edmunds, 'Brain,' vol. vii, p. 103; Obersteiner, 'Wien. Med. Jahrb.,' Bd. iii, 1879; Lochner, 'Bayer. Ärztlich. Int.-bl.,' 1875, No. 42; Fromüller, 'Memorabilien,' 1870, No. 12. In the last, described further on, softening was found thirty-two hours after the injury.

† Fischer, 'Deut. Zeitschr. f. Chirurg.,' 1883, Bd. xix.

It is therefore neither practicable nor necessary to do more, in this place, than to describe their general characters. According to the differences in course, we may divide them into three classes. (1) Those in which the injury causes immediate and severe paralysis. (2) Those in which there are at first either no symptoms or only trifling disturbance of function, but in which grave symptoms come on a few days or weeks after the injury. (3) Those in which there are no early symptoms, or only slight and transient disturbance, but at the end of one or several months symptoms gradually come on, often such as indicate disease of some definite system of structure of the cord, degenerative in nature.

The first class, in which the injury causes instant and considerable disturbance of function, includes the cases in which the cord is directly injured, and also some in which there is no visible sign of damage if the patient dies within a few days. In the latter, the mechanical influence has apparently abolished the function of the nerve-elements. It is probable that, if such patients lived longer, either quick recovery or local softening would follow. It is an interesting fact that concussion may thus derange function. The absolute integrity of structure on microscopical examination shows that the result is not due to any minute vascular lesion. The effect has been compared by Reynolds, not inaptly, to the demagnetisation of iron by a blow. Doubtless the influence is exerted on the molecular nutrition of the nerve-elements, and the possibility of recovery, or the subsequent structural disintegration, depends upon the degree of nutritional damage.

The symptoms in these cases of severe and immediate effect are generally those of complete impairment of function. When the injury is direct and partial, such as a hemisection by an incised wound, or a partial bruise of the cord by a spiculum of bone being driven against it (as in the case mentioned at p. 132), the effect may be a partial (*e.g.* one-sided) derangement of function, but in most other cases there is abolition of all the conducting functions at the level of the injury. There is complete paraplegia, motor and sensory, with loss of power over the sphincters. The symptoms are thus those of a total transverse lesion at the affected level (see p. 154). Loss of consciousness is occasionally produced by an injury which does not directly involve the head, and vomiting is very common at the outset. The cases of direct injury often run a severe and rapid course; the tendency to trophic changes in the skin, and to the occurrence of cystitis and pyelo-nephritis, is very great. Œdema of vaso-motor origin, and effusion into joints, may be present in the early stage. If the cord has been directly injured in its whole thickness survival beyond a fortnight is rare. If the cord is damaged indirectly, with secondary softening—in what may be termed concussion-myelitis—death occurs less rapidly, but many patients die at the end of four or six weeks, while in those who survive the first two months slow recovery commonly occurs. Even in such cases death sometimes occurs very

quickly, but it is possible that there is then a laceration of the cord. A man, whose case has been recorded by Fromüller, was struck on the back, at the level of the third dorsal, by a heavy beam, and had loss of motion and sensation up to the level of the nipples. The palsy of the muscles of respiration increased, and he died from asphyxia at the end of thirty-two hours. At the spot struck the cord was reduced to a pulp for $3\frac{1}{2}$ cm., without any hæmorrhage.

In cases of the second class, initial symptoms are absent or slight. After the accident, the sufferer may not imagine himself injured, may be able to walk some distance without inconvenience. In other instances there is tingling in the legs, or in all the limbs, immediately after the concussion, sometimes with some weakness of the limbs, more often with a feeling of stiffness. In the course of a few days graver symptoms come on, usually attended with spinal pain and tenderness, sometimes with some stiffness of the back, and often with pyrexia. Tingling in the limbs increases or develops, and is accompanied by weakness, which often goes on to complete paralysis in the course of a week or fortnight. Sometimes there is also loss of sensation, but less commonly than in the cases in which the symptoms come on immediately. There is often pain in the limbs, and sometimes hyperæsthesia. The symptoms vary in their character and distribution, according to the position of the morbid process and its extent. In some cases, in which the dorsal region is most affected, the symptoms are those of simple paraplegia, usually with spasm, and sometimes with early contraction of the muscles. In other cases, in which the grey matter of the enlargements suffers, there is scattered muscular atrophy in the limbs, often with indications of the degenerative reaction. Tremor in the limbs is a conspicuous symptom in some cases. A girdle-pain, or sense of constriction in the limbs, is very common. The sphincters generally suffer, and the tendency to trophic changes is usually strong. The character and course of the symptoms is that of a subacute myelitis, and the meagre facts of morbid anatomy leave no doubt that this is the common lesion. Thus a lady was severely shaken in a railway collision. She seemed, immediately after the accident, to have suffered no injury, but in a few days paraplegia developed, and from its consequences she died six weeks after the accident. Throughout the dorsal region of the cord, I found indications of subacute myelitis, chiefly in the white columns, varying in its extent in different regions, but, in most parts, considerable in the pyramidal tracts. The symptoms in these cases are sometimes unilateral. A man, driving under a low archway, leaned back to save his head, and pressed his spine suddenly against the sharp edge of the seat back. He felt but little immediate effect, but in the course of two or three days complete motor palsy of the left leg came on, accompanied by hyperæsthesia, but without any loss of sensation either in that leg or in the other. Power began slowly to return at the end of three months. Again, a clergyman was thrown from his horse,

and there was immediately sufficient weakness of the legs to prevent him from walking; this subsequently increased, so that, at the end of a fortnight the right leg was completely paralysed, while the left retained considerable power. There was a girdle-pain at the level of the umbilicus, and a bedsore formed, but he slowly improved, and regained the power of standing at the end of eight months.

When the damage involves the grey matter, the extent of the muscular wasting varies greatly. It seldom affects both arms and legs, but it is usually irregular in distribution, sometimes widespread, sometimes limited. Thus a young man fell from a horse and pitched on the head. He was stunned, and on recovering consciousness about two hours later, felt "pins and needles" in both hands, and pain in the back, followed by swelling of the neck and difficulty in moving it. The tingling ceased, but was followed by a sense of oppression about the shoulders, and persistent pain in the cervical region of the spine. When I saw him, two months later, there was some weakness of the right arm and wasting of the two outer interossei and of that part of the long extensor which acts on the two outer fingers, with loss of faradaic and preservation of voltaic irritability.

In cases which survive the acute stage, there is usually slow improvement which is often ultimately very great. In many cases recovery is incomplete, but improvement goes on for years, and the ultimate degree attained is commonly much greater than in cases of corresponding character and severity due to other causes than injury. But some permanent symptoms are often left when the initial derangement of function has been severe, and has lasted for some time. Thus a heavy weight fell upon a man's back; he was unconscious for two days, and at the end of that time the legs were completely paralysed, and continued so for three months, with retention of urine. Then improvement commenced, in the left leg before the right, and in the latter some spasm developed. At the end of six months he could walk across the room on crutches. Improvement continued, but at the end of two years his condition became stationary, and when I saw him, six years after the accident, he could only walk half a mile; the right leg was still weak in all parts, and a foot-clonus was present in each leg, greater in the right than in the left.

In the cases of the third class, chronic symptoms slowly follow an injury at an interval usually of some months, and the cases have the aspect less of a traumatic lesion of the cord, than of a primary cord disease, the relation of which to the injury is rather an inference from the sequence, coupled with the absence of other causes, than an obtrusive fact, as in the cases of the first and second classes. The symptoms are usually those of a definite system-disease of the cord, less frequently those of a disseminated chronic myelitis of irregular distribution. Primary spastic paraplegia, locomotor ataxy, and progressive muscular atrophy, are the diseases which most frequently thus result. The fact that an injury is occasionally the cause of these

maladies has been already mentioned in the account of their etiology, and the symptoms of such cases so far resemble those that are due to other causes that it is not necessary here to add anything to their history as already given. We must assume that the shock to the nerve-elements causes a slow perversion of nutrition, which is only manifested by disturbance of function when it has gradually attained a certain degree. Occasionally other causes co-operate, producing a tendency to degeneration. Thus previous syphilis, or neurotic inheritance, can sometimes be traced, and it is a reasonable assumption that the predisposition thus arising may assist in rendering the traumatic influence efficient, or in augmenting its effects.

The consequences of concussion, which we have considered as occurring in separate form, are also sometimes combined. Thus an actual and immediate lesion of the cord may be combined with an early and severe increase in the symptoms from the development of sub-acute myelitis in other parts. The traumatic inflammation at the spot injured often attains an intense degree, and runs a severe course, in consequence of the influence which may give rise to such inflammation apart from a positive lesion; and, in both cases, the damage to the cord may be perpetuated by a degenerative tendency, aided, in many cases, by the additional predisposition just mentioned. It is very common, for instance, for an injury to the cord, in those who have had syphilis, to cause acute symptoms which subside, but not perfectly, and the residual disturbance of function may persist, and even increase in the course of years, in consequence of the degenerative tendency.

One other symptom remains to be considered, which is common to all forms of traumatic lesion of the cord, and is often severe and persistent when other symptoms are slight,—spinal pain. It may continue for years after other symptoms have ceased, and probably depends on a neuralgic state of the nerves of the membranes, or of those of the vertebral column. Often, probably, the pain depends on the nerves of the joints and ligaments of the spine. It thus appears to be essentially a traumatic spinal neuralgia. The pain may be felt at one or more spots; when severe, it often extends through a considerable length of the spine, and sometimes passes up to the occiput. It is occasionally referred to the sacrum, and may there have the character of a sense of weight or more vague discomfort. The pain is associated with tenderness of the spine, usually deep-seated, chiefly developed at the injured part, but sometimes present also at other spots.

The cause of traumatic lesions of the cord often acts also on the brain. A cerebral lesion may occur from the violence which affects the cord, and cerebral symptoms then coexist with those of the spinal lesion, and may mask the latter during the early stage. More common, however, is functional disturbance of the brain, the result partly, perhaps, of the physical concussion, but chiefly of the mental

shock, which a serious accident necessarily causes. The resulting condition is favorable to the development and persistence of subjective sensory symptoms. Attention, maintained by concern, has a powerful intensifying influence on all forms of nerve-pain, and certainly aids in keeping up the pain in the back, and even the local tenderness, which follows injuries to the spine. So marked is the influence of "nervousness" on the subjective symptoms, that it has been even maintained that, in a large number of cases of concussion of the spine, the symptoms are of hysterical origin.* "Hypochondriasis" would, perhaps, be a more accurate designation for the condition in most cases in which mental influence plays a distinct part in the development or maintenance of the symptoms. But, on the other hand, it is necessary to avoid the danger of over-estimating the effect of mental influence, and of regarding as entirely due to this, symptoms which are real, and are merely intensified by attention. The danger is especially great in cases of railway injuries, concerning which an unbiassed judgment is not easy to secure, and in which, when objective symptoms are absent, it is easy to minimise suffering, and attribute too much to the mental condition. The sinister influence of litigation on the intellect may be traced very widely. I believe that it is rare for symptoms to be purely mental. It is often asserted by those employed for railway companies, that subjective symptoms quickly subside when the sufferer's "claims" are settled, but in a good many individuals whom I have had an opportunity of observing long after they had received their "damages" (as the expression curiously runs) this subsidence had not occurred, and even the "sovereign balm" of substantial compensation has appeared to do very little for the relief of the sufferer.

DIAGNOSIS.—The chief points in the diagnosis of traumatic lesion to the cord have been already incidentally considered. Immediate symptoms may be due to laceration, hæmorrhage, or to simple concussion, and the diagnosis between these is not always possible at first. If there are immediate symptoms of a partial lesion, these indicate direct injury, while the rapid subsidence of the disturbance of function renders simple concussion probable, and excludes any considerable direct injury. The later development of paralysis indicates myelitis, unless there is evidence of considerable irritation of the nerve-roots at a certain level, which suggests inflammation outside the cord, and perhaps even outside the dura mater. The greatest diagnostic difficulty is presented by the cases just mentioned, in which the symptoms are subjective, and anxious attention has been long given to the local discomfort. It is important to search for, and to give due weight to, any symptoms beyond the simple spinal pain. Slight "tingling" or "creeping" sensations may be of purely functional origin, but a persistent sensation of "pins and needles" rarely

* Putnam, 'Boston Med. and Surgical Journal,' 1883, Sept. 6th.

is of that nature. A definite sense of constriction is also strongly suggestive of organic disease, and so is a well-marked difference in the power of the muscles on the two sides. The latter is of least significance if the excess is slight, general, and on the right side,—of much greater significance if the diminution is partial, and affects only certain groups of muscles, such as the flexors of the hip and knee, or the peronci. Any impairment of power over the bladder or rectum is of great diagnostic importance; loss of sexual power, on the other hand, is of little value, since this function is readily depressed by mental anxiety and preoccupation. A slight change in reflex action is most significant when it is partial. A foot-clonus, or rectus-clonus, is strong presumptive evidence of organic mischief; a slight excess of the knee-jerk is of little value; although it probably always indicates some changes in the nutrition of the spinal cord, it does not render structural disease even probable. In all cases it should be remembered that the absence of any common symptom is of far slighter significance, as evidence of integrity of the cord, than is the presence of that symptom as evidence of disease. It may seem superfluous to insist on a consideration so elementary, but it is still possible, as experience proves, for a medical witness to assert in a court of law that a claimant's spinal cord cannot have been injured because his legs are not wasted.

PROGNOSIS.—Immediately after an accident a cautious prognosis should be given, even if the symptoms are slight, on account of the possibility that grave disturbance may develop in the course of a few days. In developed cases the prognosis must, in general, be guided by the same considerations as those which determine our estimation of the probable course of symptoms of similar character and severity due to spontaneous myelitis. To this there are, however, two general exceptions. First, the danger of death, if any exists, is greater in traumatic cases than in others. Secondly, if there is no danger to life, or such danger has passed, the prospect of improvement is distinctly greater than in a case of similar features but of non-traumatic origin. If the symptoms are slight or moderate in degree, approximate recovery may be anticipated. Often, however, the recovery, although approximate, is not perfect. The patient is never quite as strong, quite as capable of exertion, as before the injury. A cautious prognosis should be given whenever there is the late and gradual onset or increase of symptoms that suggests a degenerative process. Such degeneration presents far less tendency to arrest or subsidence than do the earlier lesions.

TREATMENT.—In all cases in which spinal symptoms are present immediately after an injury, however slight those symptoms may be, absolute rest for three or four days should be insisted on. This is necessary on account of the secondary inflammation which, as we have seen, so often occurs. The treatment of developed symptoms must be

conducted on the same general principles as in cases of myelitis; the details need not be here repeated. If there is muscular wasting it is important that the nutrition of the muscles should be maintained by electrical stimulation, since a very considerable amount of ultimate recovery may be anticipated, and it is important to keep the muscular tissue, as far as possible, in a condition to respond to the nervous power when this returns. If there is reason to believe that there is inflammation of the membranes, or inflammatory effusion outside the cord, compressing it, mercury may be given, but this condition is probably much more rare than might be anticipated. The influence of mercury on inflammation of the substance of the cord is doubtful. The degenerative sequelæ of injuries to the cord need the same treatment as the similar degenerations that occur apart from traumatic influences.

The treatment of the neuralgic condition of spinal pain and tenderness, which so often succeeds injury, is frequently difficult. Counter-irritation is often useful, either by the actual cautery, blisters, iodine, or repeated sinapisms. Of sedatives Indian hemp is most effective, next to morphia, which should be used as seldom as possible. Hypodermic injections of cocain may be tried. When all active mischief is over, and the pain has become purely neuralgic, it is often necessary to encourage the patient to neglect it in some degree, and to exert himself in spite of it, while avoiding whatever increases it in considerable degree and for a considerable time.

FUNCTIONAL AND NUTRITIONAL DISEASES.

FUNCTIONAL DISEASES.

Very little is known, though much is heard, of functional diseases of the spinal cord. It is indeed open to doubt whether there are any morbid states which can accurately be thus designated. Hysterical paraplegia is often regarded as a functional affection of the cord, because the symptoms have the same distribution as those of organic diseases of the cord, but a little consideration will show that, in a case of purely hysterical paraplegia, the morbid functional condition is cerebral; the brain-centres which act on the legs are at fault, but the condition of the functions of the cord itself may be absolutely normal. The spinal motor centres are in a state of inactivity, because the related cerebral centres are inactive, but this is no more a diseased condition of the cord than is its corresponding functional state during physiological rest. Hysterical paraplegia will be described, with other palsies of like origin, in the chapter on hysteria.

Isolated disturbance of functional centres in the cord does, however

sometimes occur. It is seen in the conditions of inhibition which are designated "reflex paralyses." Such palsies were once thought to be common and persistent, but it has been proved that many, and it is probable that all, cases of considerable and prolonged palsy, formerly thought to be of reflex origin, are due to organic disease, either primary in the cord or secondary to an ascending inflammation of nerves. But transient paralysis sometimes occurs which cannot be otherwise explained than as an inhibition of a spinal centre due to peripheral irritation. Such, for instance, is the curious inability to pass urine which sometimes follows an operation on the anus, the division of a fistula, for instance, or the removal of hæmorrhoids. The inability may continue absolute for several days. Transient weakness of one arm is said sometimes to follow an operation for empyema (Lepine), but the fact that the weakness may be attended by choreoid movements in both arm and leg of one side (Weill) makes it probable that the influence is exerted on a cerebral rather than on a spinal centre. Considerable paraplegia was thought to be sometimes a reflex effect of disease of the bladder or of a calculus or other organic disease in the kidney, but it is probable that such cases are always of organic nature.

Another class of cases which may be regarded as functional are those in which symptoms, commonly subjective in character, result from some morbid blood state. The conditions which most frequently have this effect are gout and diabetes. Occasionally there is definite failure of power, lasting for a few days or weeks, without objective symptoms, and passing away. But the most common symptoms from this cause are sensory and subjective,—feelings of tingling and formication in the legs, dull aching, and sometimes actual pain; this is usually transient, but occasionally continues for some days or weeks, various in position, but, in gouty cases, often felt in the heels. Such symptoms due to morbid blood states occur chiefly during the second half of life. Those of gouty origin, like other symptoms of the same class, are especially common in persons who inherit a tendency to gout but have not suffered from attacks of definite arthritis. The treatment of these symptoms is chiefly that of the blood state which causes them, but they may be to some extent relieved by sedatives, of which bromide of potassium, Indian hemp, and cimicifuga are the most effective.

NUTRITIONAL DISEASES.

Among the cases often classed as functional diseases of the spinal cord are some in which objective symptoms of deranged function, slight in degree but definite in character, persist for months, years, or for life. They occur chiefly in those in the early and middle period of adult life, and are more common in women than in men. Such symptoms are inability to walk more than a short distance without

fatigue, impaired nutrition of the legs, slight increase of myotatic irritability, often associated with pain in the back. If the history of such symptoms is traced they will generally be found to date from some definite exciting cause, from an attack of acute illness, such as typhoid fever or acute rheumatism, from pregnancy, a fall, and the like. The increase in myotatic irritability is exceedingly common; it is enough to permit the knee-jerk to be obtained by tapping the depressed patella, but there is rarely a distinct clonus,—at most only two or three jerks, quickly ceasing. We can hardly conceive that symptoms which are so persistent can depend on any mere functional derangement; it is probable that they depend upon changes in the finer nutrition of the nerve-elements, too slight to be detected by the microscope, causing a corresponding and persistent alteration of function. We have seen (p. 144) that the termination of the upper segment of the motor path is probably that structure of the cord which has least nutritional stability, and is therefore most susceptible of nutritional derangement. Hence we can understand the frequency with which there is an excess of myotatic irritability in these cases. There are probably gradations between such conditions and actual structural disease, in which minute examination reveals visible alteration. We have already had occasion to consider these nutritional changes as probably underlying the condition of arthritic muscular atrophy, and we have seen that the alterations in spinal nutrition in that disease are apparently the result of the impressions on the peripheral nerves. In women such a condition is often associated with uterine or ovarian pain, and with sacral pain, apparently of uterine origin. It is possible that the condition we have been considering is sometimes secondary to uterine pain, which acts in a manner somewhat analogous to that in which joint inflammation acts, and that this is the explanation of some of the cases in which a reflex disturbance of the functions of the cord has been supposed to be of uterine origin. The condition is often associated in women with spinal pain and tenderness, analogous to that which has been already described as secondary to injury, and which we shall have again to consider as a variety of neuralgia. Thus many cases of nutritional and so-called functional derangement of the cord are very complex in character.

It is rare for the indications of nutritional disturbance of the cord to pass away altogether when they have once become established. The treatment of this condition is first the removal of any present cause of depression of function, such as the diminution of uterine or spinal pain; secondly, the improvement of the general health and the avoidance of over-fatigue; thirdly, the administration of nervine tonics, such as arsenic, quinine, and strychnia. A course of massage-treatment is often of distinct service.

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